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HÆMORRHAGE, ANÆMIA AND JAUNDICE IN THE NEW-BORN.

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THE object of this paper is to describe the commoner serious diseases of the new-born characterized by hæmorrhage, anæmia and jaundice; to discuss the causation of these diseases, laying special emphasis upon the part played by hepatic insufficiency in their production; and to plead for the more frequent use of blood transfusion in their treatment.

The clinical material consists of all the infants presenting one or more of these symptoms in serious form who were born at the Women's Hospital, Brisbane, during the fourteen-month period ending March 31, 1941. One severe case of hæmolytic anæmia occurring in 1939 has been added. The total number of patients was 27, and I was privileged to treat most of them. I am indebted to Professor G. Shedden Adam, medical superintendent of the Women's Hospital, for giving me access to the records of the others.

Classification.

As a preliminary to the classification of these diseases it must be recognized that there are three mild conditions which occur so commonly in new-born babies that they are called "physiological". These are: (i) a hæmorrhagic tendency, characterized by low plasma prothrombin level and increased bleeding and coagulation times; (ii) a tendency to the development of anæmia, (*a*) from inability of the hæmopoietic tissue to meet the demands of blood formation, or (*b*) from hæmolysis; (iii) an icteric tendency, which is almost universal and which takes the form of raised serum bilirubin level or of frank jaundice.

These three "physiological" states are met with in all degrees of severity, and when considering a large series of them one eventually comes to a point at which they must be regarded as pathological. Thus the hæmorrhagic tendency at its worst produces hæmorrhagic disease of the new-born; the mild anæmia, either from anhæmopoiesis or from hæmolysis, is exaggerated into a severe anæmia requiring drastic treatment; instead of severe "physiological" jaundice one finds the deep colour and desperate prognosis of *icterus gravis neonatorum*. It seems likely that the physiological and the corresponding pathological states are related to one another. They blend imperceptibly. In a large maternity hospital babies can be found who represent every transitional stage, from those whose latent tendencies can be discovered only by biochemical means to those with a fulminating illness which kills them in a few hours.

With the intention of confining this paper to severe cases I have omitted all borderline states and some of moderate severity, retaining only descriptions of those infants whose illnesses proved fatal and those who were ill enough to require blood transfusions. Inasmuch as one never embarks without some hesitancy on a blood transfusion for a new-born baby, only the worst cases have qualified for inclusion.

In Table I the classification is based on the hypothesis that the diseases represent exaggerations of well-known "physiological" conditions:

It so happens that, although such a classification does not exhaust all possible causes of hæmorrhage, anæmia or jaundice in new-born infants, the others are rare enough to be of academic interest only. During the period under review there were no examples of them at the Women's Hospital.

TABLE I.

Physiological Condition.	Pathological Condition.	Cases.	Deaths.
I. Hæmorrhagic tendency.	I. Hæmorrhagic disease of newborn	5	1
II. Physiological anæmia	II. Anæmia of the newborn:		
(i) Anhæmopoietic	(i) Anæmia of prematurity	3	1
(ii) Hæmolytic.	(ii) Acute hæmolytic anæmia	5	—
III. Physiological jaundice.	III. *Icterus gravis neonatorum*:		
	(i) Subacute ..	7	3
	(ii) Acute ..	7	4

Hæmorrhagic Disease of the New-Born.

Hæmorrhagic disease of the new-born, sometimes wrongly named (from a single symptom) *melena neonatorum*, is not uncommon. In addition to the five cases recorded in this paper there were seven others during the fourteen-month period in which the hæmorrhage responded to the intramuscular injection of blood; this is an average of one to every 390 births. The sexes are affected equally in this disease and bleeding usually appears on the second day. Melena is the most common symptom, with hæmatemesis next; but there may be hæmorrhage from the umbilicus, vagina or kidneys, or into the subcutaneous tissues. Capon,[1] reviewing 61 cases, found melena alone in 34, melena and hæmatemesis in 21, and hæmorrhage from other sites in six.

Condensed notes of the five severe cases are as follows:

CASE I.—Baby M., was born on February 1, 1940, approximately six weeks prematurely, and weighed four pounds four ounces. Four and a half hours after birth this baby had a sudden profuse hæmatemesis, inhaled a quantity of blood and died of asphyxia.

CASE II.—Baby McN. was born on February 16, 1940, and weighed six pounds fourteen and a half ounces. A large cephalhæmatoma was present at birth. At thirty hours of age the child began to vomit bright blood, about an ounce at a time, and within a few hours became pale, cold and restless. A blood transfusion was given at thirty-five hours, and thereafter the hæmatemesis ceased and the infant recovered.

CASE III.—Baby L. was born on April 28, 1940, and weighed ten pounds one and a half ounces. Severe melena commenced when the child was thirty hours old and large clots of fairly bright blood were passed by the rectum. The condition deteriorated rapidly. Nine hours later the hæmoglobin value had fallen to 52%. The infant was pale, cold and almost pulseless. A blood transfusion was hastily given and had a dramatic effect. There was no further bleeding and the baby made a good recovery.

CASE IV.—Baby Y. was born on August 27, 1940, and weighed five pounds fifteen ounces. Thirty-six hours after birth this baby began to pass dark blood by the rectum. The hæmoglobin value was then 134%. Vitamin *K* was given by mouth (two and a half minims every three hours) and ten cubic centimetres of maternal blood were injected intramuscularly. Next day the infant looked very much worse. Hæmorrhage from the bowel was still continuing and the hæmoglobin value had fallen to 72%—an enormous drop. A blood transfusion was given and there was no further hæmorrhage. The infant did well and the hæmoglobin was 95% on the fifteenth day.

CASE V.—Baby B. was born on March 22, 1941, and weighed seven pounds eight ounces. This baby suddenly expelled a quantity of dark blood from the rectum at the age of thirty-six hours. Ten minims of vitamin *K* were administered promptly and 20 cubic centimetres of maternal blood were injected into the thighs. In spite of this treatment the hæmorrhage was repeated three times in the next six hours and the baby became pale, cold and collapsed. The hæmoglobin value fell to 60%. A blood transfusion was then given with immediate effect and the infant recovered.

Ample evidence is available to show that the underlying cause of the bleeding in this disease is a deficiency of prothrombin in the infant's plasma.[2,3] Even healthy new-born babies have only 15% to 40% of what is considered the normal adult quantity,[2,4] and between the second and third day, when the plasma prothrombin reaches its lowest levels, the coagulation time of infant's blood is nearly half as long again as the normal adult figure.

There is reason to believe that prothrombin is formed in the placenta up to the time of birth[4] and that thereafter its manufacture takes place in the liver. Experimental injury to the liver in animals and the damage resulting from chloroform and phosphorus poisoning in man are accompanied by a pronounced decrease in plasma prothrombin content and the appearance of a hæmorrhagic tendency.[5,6,7] But the liver of the new-born infant, which suddenly finds itself responsible for producing prothrombin, has two additional handicaps to overcome at the start. One is that vitamin *K*, which seems to be essential for the synthesis of prothrombin, is not available in adequate amounts during the first few days of life.[8] It becomes plentiful only towards the end of the first week, when fresh supplies are formed by bacterial action in the intestines.[9] That the shortage of this vitamin is significant can be shown by the rapid rise that occurs in the plasma prothrombin content when vitamin *K* is administered to a new-born infant. But lack of vitamin *K* is not the only factor which limits the production of prothrombin during the first few days of life. This was illustrated by an infant described by Tocantins,[8] suffering from complete atresia of the duodenum coupled with mild hæmorrhagic disease. The blood prothrombin level was very low at first; but before the baby died it had returned to normal and the bleeding had ceased spontaneously. Meanwhile no vitamin *K* from the food could have reached the intestine, and the sterility of the gut would have prevented its synthesis. It appears, therefore, that the liver is capable, after an interval, of manufacturing prothrombin from such vitamin *K* as the fœtus has obtained from its mother before birth, even when no additional supply is available. It has been demonstrated on many occasions and in various ways that there is a relative functional insufficiency of the liver during the first few days of life, and it seems likely that this factor is partly responsible for the initial lag in the production of prothrombin.

Treatment.

The cardinal point to be borne in mind is that more blood has always been lost into the lumen of the gut than one can see on the napkin, and that for this reason only a few hours may intervene between the first hæmorrhage and the infant's death. One should keep the infant as warm and as quiet as possible, but there is no need to stop the intake of fluid by mouth. While remaining in his cot he may be given sweetened water or saline solution or colostrum obtained from the mother.

There are three ways of checking the hæmorrhage: (i) administration of vitamin *K*, (ii) intramuscular injection of blood, and (iii) blood transfusion. Vitamin *K* is chiefly important as a prophylactic. If a sufficient quantity were to be given to all mothers before confinement it is probable that all babies would be born with an adequate supply of prothrombin and hæmorrhagic disease would be unknown.[9] If vitamin *K* is given to an infant when hæmorrhage has already commenced, one has to allow several precious hours for its absorption and utilization by the liver, which means that, except in mild cases, vitamin *K* therapy is too slow.

If whole blood is given intramuscularly the injection should be made away from the napkin area to avoid infection. This form of treatment suffices in the majority of cases. Ten to twenty cubic centimetres are required. Collis[10] advises repeating the injection in three hours, whether a further hæmorrhage has occurred or not. Even so, time has to be allowed for the infant to absorb the much-needed prothrombin from the injected blood, and if the hæmorrhage has been rapid death may occur during the intervening period.

In the worst cases of hæmorrhagic disease of the new-born blood transfusion is needed if the patients are to survive. This operation provides the infant with prothrombin which it can utilize instantaneously, and at the same time it provides corpuscles and plasma to replace what has been lost. I have noticed that the small cut one makes over the saphenous vein at the ankle oozes steadily

until the cannula is in place and fresh blood is being introduced, when it quickly dries of its own accord.

The immediate restoration by blood transfusion of a blanched moribund infant is a most dramatic event. Having seen how near death these babies can be and how rapidly they recover, I feel that there is little excuse for a fatality. Disaster comes only when one under-estimates the severity of the bleeding and relies upon vitamin *K* or intramuscular injections of blood in every case.

Anæmia of Prematurity.

It is a common experience with those who have the care of premature babies to find that a number of them, particularly those weighing under three and a half pounds at birth, become anæmic when they are a few weeks old. As a rule the anæmia is only moderate. When it is more severe it effectually brings progress to a standstill and may cause so much reduction in vitality that the infant fails to survive.

Several factors play a part in the causation of this anæmia. For one thing, the customary hæmolysis of the first week tends to be greater in premature babies than in those born at full term, and secondly, they miss the extra supply of iron and copper normally provided during the last month *in utero*, thus starting life with inadequate reserves. But the third and most important factor is an exaggeration of a "physiological" inadequacy of the hæmopoietic system into a gross inability to keep pace with the infant's requirements. In the worst cases, iron, copper and liver extract have no influence upon the blood picture,[11] and the only effective treatment is blood transfusion. There were three such cases in this series.

CASE VI.—Baby R. was born on March 10, 1940, and weighed three pounds two ounces. This baby suffered from mild melena and passed a few tarry stools between the second and fourth days. The bleeding ceased spontaneously, but left him pale. After an initial fall the birth weight was regained on the fifteenth day. From that time onwards steadily increasing pallor was noticed and his progress ceased. Ten days later his weight was unchanged at three pounds two ounces. The hæmoglobin value was 50% and he was not jaundiced. A blood transfusion was given on the twenty-fifth day. His colour and vitality immediately improved and during the next ten days he gained six ounces. At the age of eight weeks he weighed four pounds four ounces and was doing well.

CASE VII.—Baby C. was born on April 25, 1940, and weighed two pounds ten ounces. This was a difficult baby from the start. The child's weight fell to two pounds five ounces and the birth weight was not passed until the thirty-third day. At the age of eight weeks the child had struggled up to three pounds eight ounces, but was growing very pale. The hæmoglobin value was 55% and no jaundice was present. Three weeks later the hæmoglobin value had fallen to 44%, despite treatment with ferrous sulphate (one-quarter of a grain three times a day). The weight had become stationary, and as the child seemed likely to die of inanition, a blood transfusion was given. This brought the hæmoglobin value up to 68%, but there was no corresponding improvement in the general condition and the baby died four days later.

CASE VIII.—Baby H. was born on April 19, 1940, and weighed three pounds eight ounces. This baby also started slowly, losing seven ounces and not overtaking the birth weight for four weeks. Physiological jaundice lasted some days, and after it had faded pallor was evident. At seven and a half weeks the hæmoglobin value was only 48% and the child's weight was four pounds five ounces. Treatment with ferrous sulphate (one-quarter of a grain three times a day) was commenced. Ten days later the hæmoglobin value was still 48%; the baby was pale and listless and had gained only two ounces in weight. A blood transfusion greatly improved the colour and vitality. The child took the feeds more eagerly, gained six and a half ounces in weight in the next ten days, and was discharged from hospital later in good health.

The histories of these three premature babies show that anæmia and failure to thrive go hand in hand. Administration of iron is without effect, but blood transfusion sometimes saves a life.

Acute Hæmolytic Anæmia.

Acute hæmolytic anæmia of the new-born, also known as *anæmia gravis neonatorum*[10] and congenital hæmolytic anæmia,[12] is a serious disease; if treatment is not given it is usually fatal. Pallor is noticed within a few days or weeks of birth, being usually preceded by jaundice, which, although not deeper than "physiological" jaundice, may be more prolonged. In some cases there is no jaundice.[10] General symptoms are not pronounced until the anæmia becomes extreme. The babies are somewhat listless, however, and may not be strong enough to suck from the breast and often fail to gain weight. The blood picture reveals rapid hæmolysis with a good attempt at regeneration. The number of red cells and the hæmoglobin value are greatly reduced, but the colour index remains normal. The number of reticulocytes is high. The spleen is enlarged and there is an excess of urobilinogen in the urine. Normoblasts and erythroblasts are generally found in the peripheral blood. The fragility of the red cells is not increased and there seems to be no connexion between this disease and familial acholuric jaundice. Babies who recover have no further trouble. In fatal cases the most striking pathological finding is the evidence of great hæmopoietic activity, both in the bone marrow and elsewhere.

Four infants at the Women's Hospital suffered from acute hæmolytic anæmia during the fourteen-month period under review, and I have added the notes of another seen in 1939, whose symptoms were particularly severe. All five babies recovered.

CASE IX.—Baby G.H. was born on April 27, 1940, and weighed seven pounds fourteen ounces. This baby was healthy at birth, but on the second day he was jaundiced and somewhat pale. By the sixth day the jaundice was fading, but he was exceedingly pale. The number of red cells was 2,250,000 per cubic millimetre and the hæmoglobin value was 40%. A few nucleated red cells were seen in the blood film. His spleen was enlarged. A blood transfusion was given and he made a good recovery, leaving hospital on the nineteenth day with a hæmoglobin value of 70%. When seen some months later he was doing well.

CASE X.—Baby M. was born on October 11, 1940, and weighed eight pounds eleven and a half ounces. This baby was healthy at birth, and physiological jaundice of moderate degree lasted from the third to the tenth day. Thereafter he exhibited gradually increasing pallor, but was otherwise healthy and was gaining in weight. The hæmoglobin value fell to 53% by the end of the second week and to 44% by the eighteenth day, notwithstanding iron therapy. By this time the baby was quite blanched and listless. After a blood transfusion his hæmoglobin value rose to 75% and he remained well.

CASE XI.—Baby K. was born on April 29, 1940, and weighed seven pounds four and a half ounces. This baby was apparently healthy while in the Women's Hospital and was discharged with his mother on the twelfth day. Five days later he became a little drowsy and vomited. His mother, noticing that he was pale and slightly yellow, brought him to the Hospital for Sick Children as an out-patient. He was faintly jaundiced, but did not look ill. The hæmoglobin value was 85%. He came back four days later looking much paler, and his hæmoglobin value was down to 62%. He still looked faintly jaundiced and had been vomiting intermittently. A blood transfusion was given, and at once he became much brighter and ceased vomiting. Next day his jaundice had almost gone; three days later the Van den Bergh test produced no reaction and the hæmoglobin value was 95%. He continued to do well.

The three babies just described made complete recoveries after a single blood transfusion. Two others caused more anxiety.

CASE XII.—Baby L. was born on September 3, 1940, and weighed seven pounds fifteen ounces. Jaundice was present from birth, and when seen on the third day the child was yellow and pale, and vomited repeatedly. An immediate blood transfusion was given, no delay being made for laboratory investigations. Next day the baby was better and the vomiting had ceased. All went well for a few days, but as the jaundice faded pallor became more noticeable, and on the tenth day vomiting recommenced. The hæmoglobin value was then 65%. A second blood transfusion was given, and again the infant ceased vomiting, and energy and appetite returned. However, by the fifteenth day the child was more anæmic than ever, although the jaundice had quite disappeared. The hæmoglobin value had fallen to 50% and the number of red cells to 2,600,000 per cubic millimetre. A third blood transfusion was given, after which recovery occurred, although the child remained somewhat pale for the next few weeks.

CASE XIII.—Baby W.K. was born on July 6, 1939, and weighed ten pounds thirteen ounces. Born healthy, this baby became jaundiced on the third day and hæmorrhage from the umbilicus began. Ten cubic centimetres of maternal blood were injected into the muscles of the leg. Next day blood was still oozing from the umbilicus and the child had become very pale. The hæmoglobin value was 60%, the red cells numbered 2,600,000 per cubic millimetre, and the bleeding and coagulation times were much prolonged. After a blood transfusion the umbilical hæmorrhage ceased. A good prognosis was given, the case being regarded as hæmorrhagic disease of the new-born. However, in spite of the fact that there was no further hæmorrhage, the infant grew steadily more pale and was found to be in the grip of a severe hæmolytic process. Further blood transfusions were required on the eighth, tenth, fourteenth and twenty-third days, and in spite of these the hæmoglobin value fell at one stage to 25%. Faint jaundice persisted throughout this period. The Van den Bergh test produced an indirect positive reaction and the number of reticulocytes was high. Nucleated red cells first appeared in blood films on the eleventh day. The spleen was enlarged and the Wassermann test failed to produce a reaction. Meanwhile the baby's general condition was fair and the weight remained steady at nine pounds ten ounces. After the fifth transfusion there was considerable improvement and the infant was discharged from hospital a week later, the hæmoglobin value being 60%. For the next four weeks the child was seen regularly as an out-patient. The jaundice had gone and a rapid gain in weight took place. However, the hæmopoietic centres were flagging, and in spite of large doses of liver extract the number of reticulocytes fell gradually from 8·0% to 0·2%. The hæmoglobin value fell slowly to 38%. A few normoblasts persisted in the blood film and the spleen remained enlarged. A sixth blood transfusion was given when the child was eight weeks old; this raised the hæmoglobin value to 68% and stimulated the output of reticulocytes. Thereafter the baby remained well and when seen a year later was in splendid condition.

The diagnosis of acute hæmolytic anæmia presents no difficulty; but the cause of the disease remains obscure, and although it may be accurate to attribute the disease to an exaggeration of the hæmolytic process common to normal babies, such reasoning is not particularly helpful. It seems probable that a primary autogenous hæmolysin is at work.

The valuable effect of blood transfusions is most striking. In the milder cases a single transfusion proves curative. In the more severe cases more than one may be required; but even so, each transfusion is followed by temporary improvement. This desirable result is not merely the consequence of restoring the lost red cells. In some way the donor's blood slows up the hæmolytic process in the baby and brings it to an end. Presumably—and this is pure hypothesis—it supplies an antihæmolysin which the infant has failed to make for itself.

Jaundice.

The occurrence of jaundice in a new-born baby sometimes raises a nice diagnostic problem, and it may occasionally be necessary to consider such conditions as malformations of the bile ducts, congenital syphilis or acute pyogenic infections associated with hepatitis. Nevertheless these diseases are rare and beyond the scope of this paper. For all practical purposes the only decision that has to be made when one is confronted by a jaundiced infant is whether the condition is merely "physiological", in which case it can be disregarded, or whether, on the other hand, it is a true *icterus gravis*, in which case immediate treatment is required. Finally, it may be something between the two, in which case the infant should be closely watched.

Before considering *icterus gravis* we must be clear about the cause of "physiological" jaundice, which arises, according to standard text books,[(1)(13)(15)] from the hæmolysis of excess red cells during the first few days of life. Now it is true that babies are generally born with a high erythrocyte count, and that during the first week they sustain an average drop of approximately 150,000 cells per cubic millimetre *per diem*. But this applies to almost every baby, whereas only one in three becomes jaundiced, and there does not seem to be any significant correlation between the rate of destruction of the red cells and the level of bilirubin in the plasma.[(15)] Some other factor must therefore be concerned, and there is good evidence to show that this other factor is the capacity of the liver to excrete bilirubin. A healthy liver is capable of dealing with a much greater quantity of bilirubin than the normal baby liberates by hæmolysis. Some of the babies suffering from hæmolytic anæmia described earlier in this paper sustained a far more rapid blood destruction; yet in the later stages of their illness, while the hæmolysis persisted, their "physiological" jaundice disappeared. These babies were a waxy white colour; but infants with "physiological" jaundice are red and yellow. The hæmolysis, which is supposed to cause their icterus, is not enough to make them pale.

The cause of "physiological" jaundice is, therefore, not so much the production of bilirubin in excess as an inability on the part of the liver to deal with it adequately. In other words, there is a state of mild hepatic insufficiency in this as in other directions.[(15)]

So long as hepatic insufficiency is limited to a delay in the elimination of bilirubin, no great harm is done, and the baby, though yellow, remains well. But this condition blends imperceptibly with a more serious state. The infant is listless and drowsy, as well as jaundiced, and he looks ill; he cannot be roused to suck from the breast or the bottle; when fed forcibly he vomits. The jaundice itself becomes much deeper, and there is usually (but not always) some pallor. The liver and spleen are enlarged. Hæmorrhagic spots may appear on the skin, and occasionally frank bleeding from the rectum or elsewhere occurs. In the worst cases the child dies within a few days, sometimes within a few hours. At autopsy degenerative changes in the polygonal cells of the liver are a conspicuous feature. This is the clinical picture of acute hepatic insufficiency of the new-born, better known from its most striking symptom as *icterus gravis*.

There were fourteen such cases at the Women's Hospital during the period under review, and I have classified them into two groups: (i) subacute *icterus gravis*, in which jaundice appeared on or after the third day and the onset of severe symptoms was delayed; (ii) acute *icterus gravis*, in which jaundice appeared either at birth or before the third day.

Subacute Icterus Gravis.

CASE XIV.—Baby H. was born on April 10, 1940, seven weeks prematurely, and weighed four pounds ten ounces. Jaundice appeared on the third day and slowly became deeper. By the end of the second week the infant was intensely jaundiced and lethargic and was not gaining in weight. The hæmoglobin value had fallen to 70%. A blood transfusion was given on the fourteenth day, and immediately the baby became more lively and took food with greater zest. The weight began to increase, the jaundice gradually faded, and the child was discharged from hospital on the twenty-fifth day, weighing five pounds one ounce and looking well.

CASE XV.—Baby S. was born on August 13, 1940, and weighed five pounds two ounces; the degree of prematurity was unknown. This baby was healthy at birth and throughout the first week. After an initial fall to four pounds ten ounces the weight rose to four pounds fifteen ounces. During the second week the child became jaundiced, and as the jaundice deepened the clinical condition declined. The baby became drowsy, refused feeds and vomited, and the weight fell rapidly to four pounds four ounces by the sixteenth day. There was no anæmia; in fact the hæmoglobin value was 112% on the sixteenth day. As the infant was fast going downhill a blood transfusion was given, but without effect. The jaundice persisted, and although the loss of weight was checked, the baby died twelve days later.

CASE XVI.—Baby C. was born on March 24, 1940, approximately seven weeks prematurely, and weighed three pounds nine ounces. After an anxious start this baby began to thrive and reached the birth weight in three weeks. Meanwhile, what had been regarded as physiological jaundice appeared on the third day and seemed to be persisting indefinitely. By the end of the seventh week the baby was still jaundiced and was pale and lethargic, although still slowly gaining in weight. The hæmoglobin value was 62%. A blood transfusion was given, with immediate improvement in colour and vitality. The jaundice at once began to fade and was gone in a week. During the same period there was a gain of ten and a half ounces in weight and the infant was discharged from hospital in good health.

This baby showed many of the features of anæmia of prematurity, but is included in this series to show how the jaundice cleared up after a blood transfusion. Anæmia is not an essential part of the picture, as Cases XV and XVII show.

CASE XVII.—Baby L. was born on September 2, 1940, four weeks prematurely, and weighed six pounds six ounces. Healthy at birth, this baby became jaundiced on the third day and did not thrive from the start. When seen on the tenth day he was deeply jaundiced, although his hæmoglobin value was 105%. He was very ill, would not suck, vomited occasionally, and did not even cry. An immediate blood transfusion was given, and from that date the jaundice gradually faded. The infant seemed better for a time, but succumbed to gastro-enteritis six days later. His jaundice had quite gone before he died.

CASE XVIII.—Baby J. was born on June 12, 1940, approximately five weeks prematurely, and weighed five pounds eight and half ounces. The child became jaundiced on the third day, and when seen on the seventh day was deeply jaundiced and looked ill. The weight had fallen to four pounds ten ounces. A blood transfusion was given, and thereafter the jaundice faded and the infant began to thrive. The birth weight was regained three weeks later, and subsequently the child was discharged from hospital in good health.

CASE XIX.—Baby H. was born on April 29, 1940, and weighed seven pounds six and a half ounces. This baby was born healthy and remained well while in the Women's Hospital, except that the physiological jaundice did not fade. When discharged from hospital on the thirteenth day the baby had overtaken the birth weight and appeared to be thriving.

Four days afterwards a local medical practitioner was called to see the infant, who had become deeply jaundiced and was vomiting. He found the child a bronze-yellow colour, slightly cyanosed and moribund. There was a large hæmorrhage on the anterior abdominal wall, and crepitations were heard at the bases of the lungs. Death occurred next day.

The last baby in this series was extremely unfortunate in developing obstructive jaundice after she seemed to be recovering. This is her story:

CASE XX.—Baby S. was born on December 31, 1940, two weeks prematurely, and weighed six pounds one ounce. She left hospital at the mother's risk on the twelfth day, weighing five pounds fourteen ounces, and slightly jaundiced, but in fair general condition. The jaundice had appeared on the third day and had been persistent, but not deep. Ten days later a pale, yellow, emaciated baby was admitted to the Hospital for Sick Children because of persistent vomiting. There was no enlargement of liver or spleen, and the hæmoglobin value was 75%.

A blood transfusion brought the hæmoglobin value up to 102%, and simultaneously the vomiting ceased and the infant gained eight ounces in weight in the next eight days. Faint jaundice persisted. On the ninth day after the transfusion the jaundice suddenly became more intense, the motions lost their colour, bile appeared in the urine, and the liver became enlarged and hard. The serum gave an immediate direct Van den Bergh reaction. The baby's condition deteriorated and she became pale. Five days later, when the hæmoglobin value had fallen to 59%, a second blood transfusion was given. After that she remained fairly well except that the obstructive jaundice persisted.

She is still in hospital at the time of writing, deeply jaundiced. The motions are quite colourless and her liver is enlarged and hard. Although much underweight, she is still gaining slowly, but is unlikely to live many months.

Six of the seven babies just described were premature. All became jaundiced on or after the third day, and the special characteristic of the jaundice was its persistence. This distinguished it clinically from simple "physiological" jaundice. Other symptoms accompanied the jaundice; most of the babies were drowsy and listless, refusing to suck and vomiting what food was forced on them. They were in fact definitely unwell. Anæmia was not constant; two babies had hæmoglobin values of more than 100% when the disease was at its height. In other cases apparently an active hæmolytic process was at work. Two infants did not show symptoms until the third week, and although they were slightly jaundiced they left the Women's Hospital otherwise well. From their subsequent history one can say emphatically that new-born babies must be kept under strict supervision until jaundice has disappeared, however harmless it may seem.

Acute Icterus Gravis.

Acute *icterus gravis*, in which deep jaundice appears at birth or before the third day, is one of the most fatal diseases that affect the new-born. There were seven babies in this series; four died, two made partial recoveries and only one was completely cured. I shall briefly describe the four fatal cases.

CASE XXI.—Baby B. was born on March 9, 1941, and weighed seven pounds six ounces. This baby became jaundiced on the second day, and the depth of the jaundice steadily increased. The clinical condition deteriorated rapidly. By the ninth day the child was a deep yellow colour, drowsy, vomiting and losing weight. The hæmoglobin value was 128%. On the tenth day hæmorrhagic spots appeared on the buttocks and a large tarry motion was passed. Death occurred a few hours later.

CASE XXII.—Baby H. was born on April 9, 1940, and weighed eight pounds fourteen ounces. She was jaundiced on her first day and swiftly went downhill. Anæmia accompanied a fast-increasing jaundice. By the third day the hæmoglobin value was only 67% and the red cells numbered 3,000,000 per cubic millimetre. Numerous erythroblasts were present in the blood film. The baby had a severe hæmoptysis and died the same evening.

CASE XXIII.—Baby M. was born on March 4, 1941, and weighed eight pounds four ounces. This baby was slightly jaundiced at birth and much more so on the second day. As in the previous case, severe anæmia accompanied the jaundice. By the seventh day the hæmoglobin value had fallen to 32% and the infant was deeply jaundiced and desperately ill. A blood transfusion was then given, but the baby died three hours later.

CASE XXIV.—Baby E. was born on December 5, 1940, and weighed ten pounds thirteen ounces. This infant was faintly jaundiced at birth and became ill with surprising speed. When six hours old the child was quite yellow and the hæmoglobin value had fallen to 60%. A massive blood transfusion was given two hours later by the drip method. There was a slight temporary improvement, but the baby relapsed and died when twenty-three hours old.

I doubt if any form of therapy could have saved the baby in Case XXIV, but it is possible that early blood transfusions might have helped the others.

Acute *icterus gravis* differs from all the other diseases described in this paper, in that recovery, even when it occurs, has a tendency to be incomplete. There is a particular risk that the liver and the brain will suffer permanent damage.

The livers of infants who die in the acute stage testify to the severity of the damaging process. They are much enlarged and show fatty changes, with swelling and vacuolation of the polygonal cells and areas of necrosis. The Kupffer cells are usually swollen with hæmosiderin. As the acute stage subsides an irregular fibrosis appears among the surviving liver cells. Even in cases in which recovery has been thought complete, impaired hepatic function may be discovered some years afterwards.[18] In other instances sufficient damage is done to the liver to affect the subsequent development of the child, so that he fails to thrive and dies eventually from intercurrent infection. Case XXV is an example.

CASE XXV.—Baby G.W. was born on July 14, 1940, and weighed seven pounds six ounces. At birth this baby had ecchymoses on the face, and the stump of the cord was oozing. The bleeding soon ceased, but jaundice appeared on the first day and persisted. At the age of sixteen days he was transferred to the Hospital for Sick Children, deeply jaundiced and weighing only six pounds three ounces. He was drowsy and looked ill, and his liver was enlarged. The hæmoglobin value was 90%, the red cells numbered 5,000,000 per cubic millimetre, and many nucleated red cells were present in the blood film. The bleeding and coagulation times were greatly prolonged. The Wassermann test produced no reaction. Bile was present in the stools.

A week later the infant's weight had fallen to five pounds thirteen ounces and he was as jaundiced as ever. Although his hæmoglobin value was 96% he was given a blood transfusion, and from that day his condition began to improve. Two days later the bleeding and coagulation times were within normal limits and his hæmoglobin value was 122%. The jaundice began to fade and he was discharged from hospital a fortnight afterwards.

He attended frequently as an out-patient, but did not thrive. His colour remained a curious muddy tint, although

no actual jaundice was present. At the age of four months he weighed only nine pounds five ounces. Soon afterwards he contracted pneumonia and died. Autopsy revealed, in addition to bronchopneumonia, a small tough liver, showing advanced cirrhosis.

The unknown toxic agent in this disease which injures the liver is also apt to attack parts of the brain, and in about 5% of fatal cases damage to nerve cells can be demonstrated histologically. The commonest site is the lenticular nucleus, but the injury may be much more extensive. In a case recently described by Zuelzer and Bigler[138] gross pigmentation and extensive cellular damage were present in the hippocampus, basal ganglia and dentate nucleus of the cerebellum. In other infants the cerebral cortex is involved. The damaged areas of the brain are always bile-stained, and from the frequency with which the basal ganglia are involved the name "kernicterus" has been applied to this condition.

Cerebral damage also occurs in cases which are not immediately fatal. In such infants a variety of nervous sequelæ may develop, ranging from muscular weakness, incoordination and rigidity to spastic diplegia and mental defect, according to the portion of the brain involved. In Case XXVI cerebral complications appeared on the fourteenth day and were permanent.

CASE XXVI.—Baby Y.M. was born on September 2, 1940, and weighed six pounds eight ounces. Icterus appeared on the second day. When the baby was seen on the fifth day her skin was a deep orange colour. She was drowsy and vomiting and seemed very ill. The hæmoglobin value was 70%. A blood transfusion was given at once and repeated next morning. For a few days she was better and was able to cry and suck a bottle, and her vomiting ceased. She was still deeply jaundiced. The liver and spleen were enlarged and there was bile in the urine. The hæmoglobin value was 130%.

By the fourteenth day she had lost a little ground, and it was noticed for the first time that her head was retracted and the limbs were somewhat spastic. She was still very jaundiced and her hæmoglobin value was falling. A third blood transfusion was given, after which her condition improved greatly. By the time she was five weeks old her jaundice had gone and the infant was eight ounces above her birth weight; but the spasticity of her limbs and her head retraction persisted. The hæmolytic process was also not quite at an end, and her hæmoglobin value fell gradually to 60%; but after a fourth blood transfusion she was well enough to be discharged from hospital.

She was in hospital again at the age of thirteen weeks with a respiratory tract infection. Her hæmoglobin value was 75%, but she was small and thin, weighing only eight pounds. She still kept her head retracted and the spacticity of the trunk and limbs had increased. The liver and spleen were enlarged and firm. A month later she died of whooping cough and bronchopneumonia.

In the final case in this series the patient made a complete recovery.

CASE XXVII.—Baby K.W. was born on April 23, 1940, and weighed seven pounds eight ounces. Healthy at birth, this baby became jaundiced after twenty-four hours, and by the third day she was pale and deeply jaundiced, drowsy and vomiting. The hæmoglobin value had fallen to 60%. A blood transfusion caused a temporary improvement, but four days later the infant was as ill as ever. Further blood transfusions were given on the seventh, tenth, sixteenth, twenty-fifth and thirty-second days, and by this means it was found possible to maintain her hæmoglobin value in the vicinity of 70%. Each blood transfusion produced temporary alleviation of symptoms, and the baby became less drowsy and sucked more vigorously. Her weight fell for the first ten days and thereafter rose slowly, but she did not regain her birth weight until she was six weeks old.

Deep jaundice persisted throughout this period, and between the fourteenth and twenty-fourth days she had complete obstruction of the bile ducts, with white stools and a large amount of bile in the urine. Fortunately this serious complication rectified itself spontaneously.

The number of red cells varied with the hæmoglobin value, the colour index remaining at 0·9. A few nucleated red cells were found in blood films for the first fortnight only.

From the sixth week onwards the jaundice began to fade and the baby's condition improved more rapidly. A final blood transfusion was given at the end of the seventh week, and the baby was discharged from hospital ten days later. She then weighed eight pounds twelve ounces, and had a hæmoglobin value of 75%. There was no enlargement of liver or spleen, and her jaundice had disappeared. At the age of nine months she weighed nineteen and a half pounds, her hæmoglobin value was 80%, and she looked extremely well.

Icterus gravis neonatorum has always been a confusing disease to medical students and practitioners, chiefly because writers do not clearly define (i) its relationship to "physiological" jaundice, (ii) its relationship to acute hæmolytic anæmia, (iii) its relationship to hæmorrhagic disease of the new-born, (iv) its relationship to obstructive jaundice, (v) the significance of the family history, and (vi) the significance of nucleated red cells in the peripheral blood.

It is worth while discussing each of these points in turn.

Reasons have already been given for considering "physiological" jaundice as being due not to hæmolysis but to mild hepatic insufficiency, as a result of which the liver cells find themselves unable to deal with the relatively slight excess of bilirubin that is thrust upon them. This mild "physiological" hepatic insufficiency in the new-born extends to other functions than the excretion of bilirubin. Poor responses are shown to various liver function tests, and it appears probable that the synthesis of prothrombin is impaired as well. Possibly the detoxicating functions of the liver are also inadequate in the first few days of life. Whether this common mild hepatic insufficiency in the new-born is due to immaturity of the liver cells or whether it is due to the action of some autogenous toxic factor is a matter for speculation. We know, however, that it is impossible to make any clear distinction between the most profound "physiological" jaundice and the mildest type of *icterus gravis*. Therefore, as in the latter disease the damage suggests the action of a toxin, it seems likely that in the former case a similar toxin causes the mild and temporary hepatic insufficiency, which in turn gives rise to "physiological" jaundice.

Few writers on diseases of the new-born emphasize the point that hæmolysis and liver damage are two separate pathological processes. This is because they usually occur in association with one another; most babies with acute hæmolytic anæmia are jaundiced, at least at the commencement of their illness, and most babies with *icterus gravis* suffer also from hæmolytic anæmia. Thus jaundice comes to be described as one of the symptoms of hæmolytic anæmia, and anæmia is described as one of the symptoms of *icterus gravis*, and the student's confusion is understandable. But the true clinical picture is seen in those cases in which one pathological process only is exhibited. For example, the baby in Case X suffered from an acute hæmolysis which brought his hæmoglobin value down to 44% by the eighteenth day; yet his "physiological" jaundice had disappeared eight days earlier. Conversely, the babies in Cases XV, XVII and XXI were deeply jaundiced, suffering from *icterus gravis neonatorum*, yet the lowest hæmoglobin value recorded among them was 105%. Similar cases are recorded in the literature.[139] When it is realized that hæmolysis and liver damage are two separate processes, one can recognize without any confusion that both are generally combined in these sick babies, the emphasis sometimes being laid on one process and sometimes on the other. In such a case as Case XXVII it is a matter of choice whether the child is regarded as suffering from acute hæmolytic anæmia with severe liver damage or from acute *icterus gravis* with severe hæmolysis.

The occurrence of a purpuric rash is common in babies suffering from *icterus gravis*, and occasionally severe hæmorrhage may occur (vide Cases XXI and XXII). While there is no immediate connexion between jaundice and hæmorrhage in new-born babies, they both signify changes in the liver, the former reflecting damage to the liver cells and the latter the inadequate production of prothrombin. Since the latter process does not necessarily imply injury to the liver, it is not to be expected that jaundice would accompany hæmorrhagic disease of the new-born, and this is the case. On the other hand, in the case of babies with *icterus gravis* whose livers are gravely damaged, it is not surprising that the manufacture of prothrombin is interfered with and that hæmorrhage frequently ensues.

Two babies who were suffering from *icterus gravis* developed biliary obstruction during the course of their illness. In Case XX the trouble proved permanent, but in Case XXVII it cleared up spontaneously after ten days. The occurrence of biliary obstruction is shown by the sudden disappearance of bile from the stools, an increase in the jaundice, the presence of large quantities of bile in the urine and enlargement and hardening of the liver. The immediate cause is the plugging of the small intrahepatic bile ducts with bilirubin débris. This is an unfortunate complication of *icterus gravis*, but it is only an infrequent and accidental occurrence and forms no necessary part of the pathological picture. Yet in a recent text-book,[17] under the heading "Hæmolytic Anæmia and Jaundice", one finds the following passage:

> This pathological process of hæmolysis may in some cases be so acute that the bile ducts are unable to deal with the excess bile pigments excreted by the liver and become blocked. A true obstructive jaundice is then superadded (icterus gravis). The condition is recognised by bile pigment in the urine . . .

It is small wonder that students are confused!

Every text-book mentions the curious family history of *icterus gravis*, which is a dramatic tale that sticks in the memory. The first one or two babies in the family may be born healthy. The next baby has *icterus gravis*. After this every other baby born to that mother is likely to die from the same cause. Some text-books leave the reader with the impression that the disease always behaves in this manner. Sheldon[12] describes it as "familial icterus gravis", although he admits that "isolated instances also arise". The point I wish to make here is that in these days of small families "isolated instances" are the most common. Of the seven cases of acute *icterus gravis* in this series, three occurred in the absence of any previous jaundiced babies in the family. The fourth mother had had two stillborn children, the cause unknown. In the other three cases no inquiry was made.

It is not generally recognized that the presence of nucleated red cells (mainly erythroblasts) in the peripheral blood is an inconstant feature of *icterus gravis*. They were found in most, but not all, of my cases, sometimes being present only in the early stages, sometimes only in the later stages. Their presence and numbers seemed to have no bearing on prognosis. A similar inconstancy has been noticed by other writers,[13,14,15] and it must be concluded that erythroblastæmia is not therefore an essential part of the clinical picture. It is probably a well-meaning but imperfect attempt at compensatory hæmopoiesis.[16] Nevertheless the term *erythroblastosis fœtalis* has been introduced to link together three diseases of the new-born in which one usually finds erythroblasts in the blood stream during life and at autopsy in numerous areas of extramedullary hæmopoiesis. The three diseases are acute hæmolytic anæmia, *icterus gravis* and *hydrops fœtalis*. One must question the wisdom of such a title. The three diseases are undoubtedly related; but the presence of erythroblasts does not seem to be an important factor in any one of them.

Let me briefly sum up the foregoing paragraphs. Physiological jaundice and *icterus gravis* are related and probably represent different degrees of liver damage from the same toxic agent. Hæmolysis of red cells and necrosis of liver cells are two entirely separate processes, although they often occur simultaneously in the same infant. The hæmorrhagic tendency in *icterus gravis* can be attributed to liver damage. The occurrence of obstructive jaundice is entirely accidental. Too much stress should not be laid on the family history or on the presence of nucleated red cells in the blood stream.

Treatment.

It is generally accepted by pædiatricians that blood transfusion, repeated as often as necessary, is the only treatment that can claim to cure *icterus gravis neonatorum*. The case histories recorded in this paper substantiate the claim. Almost without exception, even in the case of the infants who finally failed to recover, transfusion at least conferred temporary relief. No other treatment is known to be of any avail, and the intramuscular injection of blood is useless. Vitamin *K* is of value only to check an associated hæmorrhagic tendency.

The reason for the good effect of blood transfusion is unknown. The fact that it leads to a decrease in jaundice and to the cessation of vomiting, drowsiness and general illness suggests that in some way it enables the liver cells to withstand whatever toxic factor is damaging them. This brings me to my last point. If the baby is deeply jaundiced, drowsy and vomiting, he should be given an immediate blood transfusion, however high his hæmoglobin value may be. To wait for anæmia to develop is to withhold the only treatment that can save his life.

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NUTRITIONAL ANÆMIA IN INFANCY: A REVIEW OF 277 SYDNEY INFANTS AT THE AGE OF NINE MONTHS.

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THE investigations reported in this paper were commenced because I had been unable to find any authoritative Australian statements, based on personal research, regarding the blood picture of infants at the time of changing from a purely milk diet to the mixed feeding instituted at about nine months of age. My experience in general practice was that many infants were referred from the health centres for an opinion as to why they were not

[1] Work done partly with the aid of a grant from the National Health and Medical Research Council.

progressing as expected. After the addition of iron to their diet, even though they were apparently healthy and exhibited no pallor of the conjunctivæ and lips, these children improved greatly in health.[1]

Inquiries among my fellow practitioners revealed that they were not alive to the importance of nutritional anæmia. One is apt to consider that because babies are being reared in a sunny climate and in a country with a higher standard of living than obtains in England, the findings in England will not be duplicated here in Australia. The monumental work of Dr. H. M. M. Mackay, in 1931,[(2)] covered the whole field of nutritional anæmia as applicable to England. Her report[(2)] that the morbidity rate among infants was halved by the addition of iron to their diet should provide thought for all who are interested in preventive medicine. This is the opinion also of American investigators, fuller details of whose work will be given later.

Although it is nine years since the publication of Dr. Mackay's work, I found that except in two cases no infants in this series, either full-time or premature, had been systematically given iron. However, egg yolk had been introduced into the diet in six cases when the children were aged six months or younger. No premature infants examined had had a blood examination, and only two had had extra iron added to their diet.

Many practitioners are under the impression that iron given to the nursing mother will prove useful to a breast-fed baby. Dr. Mackay states that she has no proof that iron given to a lactating mother, or to a mother during the last six weeks of pregnancy, has the slightest effect on the iron content of the infant's blood. Her opinion is based on a large series of cases, and she appears to be the first worker to have thoroughly investigated the problem.

Two children recently seen, but not taken from the series under review, provide examples of the absence of knowledge of nutritional anæmia on the part of both medical practitioners and clinic sisters.

CASE 1.—B.P., aged eight months, was brought to me not because she was ill, but because a medical friend, not her own doctor, had told the mother that premature babies were sometimes anæmic. The infant had been born six weeks prematurely by Cæsarean section, but had thrived and now weighed eighteen pounds eleven ounces when clothed; the birth weight was four pounds two ounces. Jaundice, which developed three days after birth, had lasted for two months. The sisters at the clinic to which the child was taken had been pleased with the weekly increase in weight. Examination of the blood of this apparently healthy infant revealed a hæmoglobin value of 62% (8·7 grammes per 100 cubic centimetres; a hæmoglobin value of 100% is 14 grammes of hæmoglobin per 100 cubic centimetres of blood).

Surely in this case iron therapy was indicated from an early age, if any credence is placed in the results of the painstaking research of Dr. Mackay.

CASE 2.—W.D., aged eighteen months, was referred to me nine months ago because he had not increased in weight for two months. His birth weight was five pounds seven ounces, and he was born two weeks before the expected date. He had been fed on breast milk only to the age of six months, and then orange juice and clinic emulsion had been added to his diet. (The clinic emulsion most commonly used in New South Wales consists of 50% cod liver oil emulsified with gum acacia. The vitamin *A* content is 500 international units in each cubic centimetre or 14,000 international units in each fluid ounce, and the vitamin *D* content is 50 international units in each cubic centimetre or 1,400 in each fluid ounce.) The child at the age of nine months, clothed, weighed eighteen pounds, and his blood had a hæmoglobin value of 58% (8·1 grammes of hæmoglobin per 100 cubic centimetres). Iron and ammonium citrate were given in gradually increasing doses until he was taking 12 grains three times a day. This was continued until he was twelve months old, when he weighed twenty-two pounds, clothed, and his blood had a hæmoglobin value of 82% (11·5 grammes per 100 cubic centimetres). Iron therapy was discontinued a month later by his mother, and when the child was seen by me again at the age of eighteen months the hæmoglobin value of his blood had dropped to 74% (10·4 grammes per 100 cubic centimetres), despite the fact that he was now taking a full diet containing five eggs per week.

Mackay[(2)] states that all artificially fed infants and many breast-fed infants should be treated prophylactically with iron, especially if their birth weight is low. She advises iron and ammonium citrate, one and a half grains, three times a day, to be given continuously to the age of twelve months. She states that this will improve the children's health and increase their resistance to disease.

INVESTIGATION AND ANALYSIS OF RESULTS.

My primary aim was to obtain the average values for hæmoglobin, red blood cells, colour index, and corpuscular volume among apparently healthy infants at the age of about nine months, and to set these up as an average found in Sydney in a survey of 277 infants. As the investigation progressed, however, I decided to analyse the results according to the type of feeding that had been predominantly employed. A division was made into three classes, namely: (i) breast-fed infants, (ii) infants fed on cow's milk and (iii) infants fed on "Lactogen" and "Vi-Lactogen". The number in each group appeared to be sufficient to allow of subdivision according to the periods of time for which each type of feeding had been employed. Twins, premature infants and infants receiving iron for a period of three months or more were included in the general results, but were also considered separately under their own headings.

PARTICULARS OF CASES INVESTIGATED.

Two hundred and seventy-seven infants seen at suburban health centres and at the Nestlé and Anglo-Swiss "Lactogen" Clinic in Sydney were investigated. The infants were examined as far as possible at about the ninth month (the average age of all infants examined was nine months), and an attempt was made to determine the presence or otherwise of nutritional anæmia in Sydney amongst apparently healthy infants fed according to present-day methods.

The cases were selected, in so far as only those children were examined whose mothers were interested in clinic methods and in the investigation being carried out. The children were all taken from working-class families, the average income being £237 6s. *per annum*. More than 25% of the parents, either mother or father, had been unemployed for periods of more than one year during the past eight years.

The average weight of the infants examined, after 12 ounces had been deducted for clothing, was as follows: males, 19·78 ± 0·2 pounds; females, 18·94 ± 0·17 pounds. These figures are slightly higher than those obtained by Dr. F. W. Clements[(4)] in 1933. His figures were as follows: males, 18·9 ± 0·043 pounds; females, 18·36 ± 0·04 pounds. From this it appears that my subjects have been selected for me—that is, the sisters at the clinics have shown me the babies of which they are proudest.

The average birth weight of the infants in this series was seven pounds six ounces. The measurements of the infants were as follows:

Average Length:

Male	27·75 ± 0·102	inches.
Female	27·25 ± 0·104	inches.
Difference	0·5	inch.

Circumference of Head:

Male	18·03 ± 0·034	inches.
Female	17·63 ± 0·05	inches.
Difference	0·4	inch.

Circumference of Chest:

Male	18·15 ± 0·075	inches.
Female	17·69 ± 0·075	inches.
Difference	0·46	inch.

Circumference of Abdomen:

Male	17·95 ± 0·085	inches.
Female	17·60 ± 0·086	inches.
Difference	0·35	inch.

The average age of the mothers was twenty-eight years. The average hæmoglobin content of their blood was 83·3% ± 0·6% (11·7 grammes per 100 cubic centimetres). Throughout this investigation a hæmoglobinometer was

[1] It had been my intention to review these cases when the children were aged eighteen months; but owing to serious illness and later to the outbreak of war, this was impossible. At the conclusion of the war the investigation will be completed.

used with 100% corresponding to 14 grammes of hæmoglobin per 100 cubic centimetres of blood. The figures for nurses at the Royal Prince Alfred Hospital (as supplied by Thomson, 1940[42]) show a much higher hæmoglobin level for single women than is found among the mothers in my series. Of 573 nurses, 521 had a hæmoglobin level above 12 grammes per 100 cubic centimetres (85·7%), 131 having a hæmoglobin level above 14 grammes (100%). It must be noted, however, that Thomson's results were obtained from a group who were on a higher social plane and enjoyed a higher standard of living.

Hæmoglobin Level at the Age of Nine Months.

Reviewing the results obtained in this investigation, one is faced with the problem of deciding what shall be considered normal for the age and what shall be considered to constitute anæmia. A search of the literature reveals practically no Australian figures. Kincaid,[32] in 1935, estimated the hæmoglobin content of the blood of 41 babies, but gave no actual figures or ages and drew no definite conclusions. She stated that of 19 infants not given extra iron, four had hæmoglobin values below 70%, whilst of 21 given iron none had hæmoglobin values below that figure. Jennings, of New Zealand,[37] in 1937, published a short communication on the subject. In 78 full-time breast- and bottle-fed infants he found an average hæmoglobin value at six months of age of 12 grammes per 100 cubic centimetres (86% by the standard used in my investigation). No other Australasian work can be found, so that the publication of my results may stimulate others to explore a vast and hitherto untouched field of preventive medicine in Australia.

In England and Scotland the results obtained fall short of the normal figures postulated by Mackay.[30][27][29] She considers that the normal level of hæmoglobin from six months of age to early childhood should be 86%. Davidson, Fullerton and Campbell[7] accept this figure as the normal for breast-fed infants after iron treatment. Their average among 44 infants at the age of nine months was 77·3% (100% = 13·8 grammes of hæmoglobin per 100 cubic centimetre).

Mackay[27] found that those offspring of mothers of the hospital class who had been entirely breast fed to the age of seven months had an average hæmoglobin value of 72·5%. She had reported in 1928 and 1939[28] a hæmoglobin average of 68·8% among 30 infants aged between eight and nine months, who were artificially fed and untreated with iron. Two years previously, in an investigation of 70 children, she had obtained an average of 68·2% in the same type of infant. Fullerton, in Aberdeen,[12] found among 66 infants at the age of nine months an average hæmoglobin of 73·8%. He made the observation that of 298 infants in the poorer classes in Aberdeen between the ages of nine and twenty-three months, only 13% had a hæmoglobin value of 80% or more.

American workers obtained much higher values than those found in England and Scotland. Merritt and Davidson[23] explain that this is due to the American practice of giving iron-rich food to the infant much earlier than is the case in England. By the age of eight months, practically all the infants in this series of Merritt and Davidson had had foods rich in iron (egg yolk, vegetables and fruits) for several months. Merritt and Davidson found in 20 children at the age of nine months a level of hæmoglobin of 12·9 grammes per 100 cubic centimetres (92% by the standard used in my investigation), with standard deviation of 1·3, and standard error of mean of 0·19. Appleton[2] emphasized the importance of interpreting the hæmoglobin estimations of infants in accordance with the values normal for the age of the subject rather than with those normal for the adult. He examined 17 subjects, aged between six and eleven months, and found the average to be 87% (100% = 13·8 grammes per 100 cubic centimetres). His highest and lowest figures were, respectively, 111% and 73%. Mayer[21] in his chart gives the hæmoglobin value of children aged between six and eleven months as 13·8 grammes per 100 cubic centimetres (98·6% by the standard used in my investigation). From his discussion of the subject, however, these figures appear to have been taken from Williamson,[40] whose results and methods have been criticised by many workers. Williamson's figures were based on an examination of 33 infants. More recently Kato and Emery,[20] in Chicago, made 154 estimations on infants aged between seven and twelve months, and found a mean of 10·7 grammes per 100 cubic centimetres (76·4% by the standard used in my investigation). There was a standard deviation of 1·4. Elvehjem, Peterson and Mendenhall,[38] using the Newcomer method in 91 estimations, found an average hæmoglobin value of 11·4 grammes per 100 cubic centimetres (81% by the standard used in my investigation). They excluded from their series all premature infants, twins and children obviously anæmic or receiving treatment for anæmia. They found the figures for male and female infants to be almost identical. These workers[39] consider that the normal hæmoglobin values for children aged between three and twelve months should be from 12·0 to 13·5 grammes per 100 cubic centimetres (86% to 96·4% by the standard used in my investigation), because all curves obtained by estimations after copper and iron therapy flatten at that level. Mugrage and Andresen,[34] in 18 estimations on infants aged between four and twelve months, give an average of 11·81 grammes per 100 cubic centimetres (84·4% by the standard used in my investigation), with a standard deviation of 0·65 and a standard error of mean of 0·10. Munday and co-workers[26] give figures for white and Negro infants in America. At the age of about nine months 191 white infants had an average hæmoglobin level of 11·27 grammes per 100 cubic centimetres (80% by the standard used in my investigation) and a standard error of mean of 0·07. Seventy-two Negro infants had an average hæmoglobin value of 10·81 grammes per 100 cubic centimetres (77·2% by the standard used in my investigation) and a standard error of mean of 0·14.

Canadian figures, as supplied by Usher MacDermot and Lozinski,[46] reveal the difference in hæmoglobin values as between infants taking iron or iron and copper and control infants. Their figures are as follows: control group, 11·5 grammes per 100 cubic centimetres (82% by the standard used in my investigation); group taking one and a half to three grains of iron per day, 13·2 grammes per 100 cubic centimetres (94%); group taking one and a half to three grains of iron and one-sixtieth to one-thirty-second of a grain of copper sulphate per day, 13·4 grammes per 100 cubic centimetres (95·7%). Their series comprised 233 infants, and they made the interesting observation that, in regard to infections, the group taking iron had a slight advantage and the group taking iron and copper had a greater advantage over the controls. In regard to mortality rate there was a moderate advantage in the group taking iron, but a striking one in the group taking iron and copper, especially in regard to whooping cough.

Faxen, of Sweden,[11] obtained high hæmoglobin values for his series of normal infants. He gives a value of 13·7 grammes per 100 cubic centimetres (98% by the standard used in my investigation) as the mean for infants at the age of five months.

In my series of cases, among a comparable class of infants, but of course in a different country under different living conditions, the hæmoglobin values were higher than Mackay's or Kato and Emery's, and were only 3·8% lower than those which Mackay regards as normal. My figures were as follows:

Male (149 Subjects).

Mean	83·13% hæmoglobin value (11·6 grm. per 100 c.cm.)
Standard deviation	8·87%
Standard error of mean	0·72%
Therefore mean =	83·13% ± 0·72%

Female (128 Subjects).

Mean	81·3% hæmoglobin value (11·4 grm. per 100 c.cm.)
Standard deviation	8·8%
Standard error of mean	0·78%
Therefore mean =	81·3% ± 0·78%

Difference between male and female	1·83%
Standard error of difference	1·06

Therefore difference = 1·7 times its standard error.

Statistically this difference is not significant, and is probably due to chance sampling. Thus it may be said that for the sample investigated there is no significant difference between male and female hæmoglobin figures. In these circumstances it is permissible to group males and females together for comparison purposes.

Hæmoglobin Value of All Infants.

Mean = 82·2% (11·5 grammes per 100 cubic centimetres).
Standard error of mean = 0·48%.

Technique.

The hæmoglobin value was estimated as follows:

A Heilige normal hæmometer was used, consisting of glass prisms of the colour standard, one on either side of the tube containing the blood solution under test. On the scale 100% is equivalent to 14 grammes of hæmoglobin per 100 cubic centimetres of blood. Twenty cubic millimetres of blood taken from a prick made on the ball of the thumb, after the part had been cleansed with ether and allowed to dry, were added to N/10 hydrochloric acid in the centre tube, and more N/10 hydrochloric acid was added until the colours were matched.

The results were read in percentages. They have been converted to grammes also, as the various workers differ in their method of giving their results.

Discussion.

The 100% standard varies in Sydney hospitals from 13·8 to 17·3 grammes of hæmoglobin per 100 cubic centimetres. As far back as 1916 Williamson[69] stated that the hæmoglobinometer readings should be standardized in absolute terms, most conveniently as grammes of hæmoglobin per 100 cubic centimetres of blood. If results were always given in a fixed unit such as grammes and never in a variable unit such as percentage, then all practitioners would soon learn to interpret their results accordingly. The interpretation of the results as obtained at present in Sydney hospitals must cause considerable difficulty. A reading of 11·6 grammes per 100 cubic centimetres would be interpreted at one hospital as 84%, whilst at another Sydney hospital the result would be given as 67%. As both these hospitals are teaching hospitals, one dealing with adults mainly and other with children, one can imagine the confusion caused to the honorary medical staff, resident medical staff, students and outside practitioners, unless the 100% standard of each hospital were known.

The figures for hæmoglobin obtained in this investigation compare very favourably with figures obtained in other countries. The mean for Sydney children is 10% higher than Mackay's London figures, and 5·8% higher than Kato and Emery's Chicago figures. Other American investigators[3,13,24,25,46] give results approximating to those found in my series. My values are, however, very much lower than Appleton's and Faxen's. Faxen's average differs widely from the English average; it was taken from children who had been carefully selected and who had to conform to certain conditions as regards maturity at birth, subsequent diet and general health. The mean hæmoglobin percentage of 82·2 obtained by me would seem to indicate that the incidence of nutritional anæmia in Sydney is not high, if one accepts as correct Mackay's standard of the normal for the age. Whether the mean hæmoglobin value could be raised in this country by the administration of iron from an early age is a matter which needs further investigation. If the addition of iron to the diet were to raise the hæmoglobin value to a higher level, then the presence of a deficiency anæmia would be established.[35] Several investigators[3,11,21,32,45,49] consider that the figure to be aimed at should be much higher than that obtained by me in this investigation.

There is a wide divergence of opinion as to whether the anæmia present in infancy is pathological or physiological. Price[41] states that anæmia of a moderate degree is present in practically all infants, whether breast fed or artificially fed, and that it may become severe, especially in twins or premature babies, or when suckling is prolonged beyond the normal time. He states that it disappears spontaneously on the adoption of a mixed diet, but adds that there are very few babies, whether breast fed or bottle fed, who are not improved in health by the prescription of iron. Parsons[37] thinks that the 84% to which the hæmoglobin value can be raised by the administration of iron should be regarded as the normal at the age of six to nine months, rather than the usual figure of 70% to 75% obtained without iron. He maintains that the statement of Castle and Minot,[12] that iron only replaces a deficiency and never produces a *polycythæmia vera*, justifies the procedure, for if the hæmoglobin value can be raised by this method, the existence of a deficency is proved. A further justification is the observation of Mackay,[33] that babies given iron show an increased freedom from infections.

Whether Sydney infants need iron added to their diet soon after birth, even though they have at the age of nine months an average hæmoglobin value of 82·2%, is a matter which needs further investigation. There were, however, 21 infants (7·6% of the whole series) who had a hæmoglobin value of less than 72%. This percentage was taken, as it was 10% below the mean of the series. Infants whose hæmoglobin value was below this figure could be definitely considered anæmic. Nine of these were premature and at birth weighed less than six pounds. The cause of the anæmia was either prematurity, low birth weight, or both conditions combined. Hugounenq[15] showed that two-thirds of the iron present in the body at full term were laid down in the last three months of intrauterine life. Mackay,[33] however, demonstrates a strong correlation between birth weight and hæmoglobin level from one to twelve months of age, irrespective of prematurity. She state that the higher the birth weight, the higher, on the average, is likely to be the hæmoglobin level from the age of one month and during most of the first year of life. The other twelve infants (4·3% of the series) will be considered in detail.

CASE 208.—H.F., aged eight and three-quarter months, had been fed on cow's milk from the age of ten days. The birth weight was six pounds and the weight at the time of examination was fifteen pounds nine ounces (clothed). The hæmoglobin value was 62% (8·7 grammes of hæmoglobin per 100 cubic centimetres), the red blood cells numbered 4,470,000 per cubic millimetre, the colour index was 0·8, and the corpuscular volume 22%. An examination of the mother's blood revealed a microcytic hypochromic anæmia; the hæmoglobin value was 46% (6·4 grammes of hæmoglobin per 100 cubic centimetres), the red blood cells numbered 3,760,000 per cubic millimetre, and the colour index was 0·61. Anisocytosis was present; central pallor and slight polychromasia were noticed, and the red blood cells were microcytic.

The mother's anæmia, in addition to the low birth weight, probably determined this infant's anæmia.

CASE 210.—J.W., aged ten months, had been breast fed from birth. He had been given orange juice from the age of three months. The hæmoglobin value was 69% (9·7 grammes of hæmoglobin per 100 cubic centimetres). He had had pneumonia at the age of five months.

This apparently was the cause of his present anæmia. Josephs[28] states that infection is a potent cause of anæmia.

In the other ten cases no explanation of the anæmia could be found. All the mothers had a hæmoglobin value of over 90% (12·6 grammes of hæmoglobin per 100 cubic centimetres). Four of the infants at birth weighed over nine pounds; the others had weighed between six pounds five ounces and eight pounds. None had had illnesses, apart from colds in the head, since birth. The present weight of all of them approximated to the average for the series, and all the infants appeared healthy. The three types of infant feeding were represented almost equally in these cases. Perhaps this group (3·6% of the series) represents those who, in later life, develop pathological anæmia.

Although in this investigation there was only a weak correlation between hæmoglobin value and morbidity rate, hæmoglobin value and weight of the child, hæmoglobin value and parents' wages, hæmoglobin value and age of the mother, hæmoglobin value and muscle tone of the infant, the hæmoglobin levels associated with the various types of infant feeding are nearly significant, and might have been significant if sufficient numbers had been tested. This is a line of investigation which I am sure would amply repay further research.

Hæmoglobin Value in Relation to the Type of Feeding.

The hæmoglobin value in relation to the type of feeding is shown in Table I.

TABLE I.

Type of Feeding.	Number of Children.	Mean Hæmoglobin *per Centum*.	Standard Deviation.	Standard Error.	Difference.
Breast milk.					
Up to the age of four to six months ..	25	82·2	7·2	1·44	—
Up to the age of seven to nine months ..	136	82·2	8·1	0·69	—
Cow's milk.					
From one month onward ..	16	78·1	6·8	1·7	—
From two months onward ..	10	83·0	6·0	1·9	—
From three months onward ..	25	82·6	6·6	1·33	—
From four to six months ..	25	82·6	6·6	1·33	—
"Lactogen" and "Vi-Lactogen".					
From one month onward ..	13	88·8	6·3	1·8	3·4
From two months onward ..	7	85·8	7·5	2·8	1·0
From three months onward ..	5	87·0	9·9	4·4	1·1
From four to six months ..	14	88·5	7·8	2·1	2·9
From seven to nine months ..	40	82·2	8·0	1·26	—

In general, the results for breast feeding and cow's milk agree closely with the mean for the series. The only outstanding exception is the group fed on cow's milk from the age of one month onward. The difference of 4·1% in hæmoglobin value, however, is not statistically significant, and may be due to chance sampling.

The hæmoglobin values for the group fed on "Lactogen" and "Vi-Lactogen" are higher by from 3·6% to 6·6% than the mean for the series, except when feeding with the preparations was commenced at the age period between seven and nine months. The figures for this period must be discarded, because of the short time for which the preparation had been taken.

In the fifth column, under the heading of "Difference", is given the number of times the difference is greater than its standard error. It will be noticed that the period from one month onward it is significant, whilst it is nearly significant for the period from four to six months onward. The number of infants examined is greater in these two groups than in the groups from two months onward and three months onward. It is reasonable to assume that if the numbers investigated had been greater, the general values for children fed on "Lactogen" and "Vi-Lactogen" would have been significantly higher than the general mean for the whole population. The higher average hæmoglobin level of infants fed on "Lactogen" and "Vi-Lactogen" is probably due to the added iron in these preparations.

Mackay,[25] quoting from various authors, states that the iron content of human milk is 0·00014% to 0·000717% of undried material and that that of cow's milk varies from 0·000187% to 0·000367% of undried material. Josephs[13] states that milk has an extremely low iron content, not more than one milligramme per litre and probably less. He considers that it varies with the amount of handling it receives between the cow and the analyst. He holds that breast milk likewise contains little iron, probably very little, if any, more than cow's milk, though analyses differ on this point.

Workers at the Nestlé Research Laboratory[6] state that the usually accepted figure for iron in cow's milk is 0·7 part per million, a figure in close agreement with those they have obtained over a considerable number of years. In the manufacture of "Lactogen" they add sufficient iron and ammonium citrate to give in the reconstituted food an added amount of iron equal to four parts per million. This is about double the amount usually found in human milk. On analysis of "Lactogen" they repeatedly find close to 35 parts per million of iron and, taking the average concentration of "Lactogen" (that is, the number of times the fresh milk must be concentrated to yield a dry "Lactogen") as 7·6 times, this gives an equivalent figure of 4·6 parts per million in the reconstituted food. If allowance is made for the amount of added lactose which goes into the make-up of "Lactogen", the amount of iron coming from the fresh milk (this fresh milk being assumed to contain 0·7 part per million) would be 0·63 part per million. So that if we deduct this from their analytic figure of 4·6 parts per million, we obtain the 4·0 parts per million that they originally desired to add.

Similarly, to "Vi-Lactogen", which contains larger amounts of added lactose, enough iron and ammonium citrate are added to give 4·0 parts per million, again when diluted and ready to be used. Analyses of "Vi-Lactogen" always give, within experimental error, 35 or 36 parts of iron per million. Since "Vi-Lactogen" is concentrated eight times, this means 4·4 to 4·5 parts per million in the diluted food. But, starting with a fresh milk of 0·7 part per million of iron, and allowing for the amount of lactose added in manufacture, one finds that the reconstituted "Vi-Lactogen" contains only 0·44 part per million of iron, derived from the original fresh milk used. This, incidentally, would be the calculated figure for a fresh milk modified to 1·7% protein by health centre methods. If this is deducted from their average analytical result, a figure of 4·0 parts per million is left—the theoretical amount they added.

These figures, given as grains per pint, are as follows:

For fresh milk, 0·7 part per million = 0·0061 grain per pint (equivalent to 0·036 grain of iron and ammonium citrate per pint).

For "Lactogen" reconstituted, 4·6 parts per million = 0·0401 grain per pint (equivalent to 0·24 grain of iron and ammonium citrate per pint).

For "Vi-Lactogen" reconstituted, 4·4 parts per million = 0·0384 grain per pint (equivalent to 0·23 grain of iron and ammonium citrate per pint).

If a six-weeks-old baby is fed on "Lactogen" according to the table on the tin, feeding every three hours would entail six feeds, each of two tablespoonfuls of "Lactogen" and eight tablespoonfuls of water. This means twelve tablespoonfuls of "Lactogen", each of which weighs 0·2 ounce, or 2·4 ounces of dry "Lactogen" per day. As this mixture is not at full strength, one must calculate the amount of iron from the weight of "Lactogen" used, and since "Lactogen" by analysis gives 35 parts per million of iron, in 2·4 ounces there is (2·4 × 35 ÷ 1,000,000), or 0·000084 ounce. Since there are 437·5 grains in one ounce, this means that each day the infant will receive 0·03675 grain of iron (equivalent to 0·22 grain of iron and ammonium citrate).

If the patient is fed every four hours—that is, if he receives five feeds at the age of six weeks—he will be given two tablespoonfuls and one and a half teaspoonfuls of "Lactogen" in addition to nine and a half tablespoonfuls of water per feed, or, in all, 11 tablespoonfuls and three and a half teaspoonfuls per day; this is fairly close to the 12 tablespoonfuls that have just been considered, and consequently one can state that the infant will again receive 0·03675 grain of iron (equivalent to 0·22 grain of iron and ammonium citrate).

At the age of nine months each feed will comprise six tablespoonfuls of dry "Lactogen" and 16 tablespoonfuls of water, and on the assumption that five feeds per day are given, this will amount to 30 tablespoonfuls of powder and 80 tablespoonfuls of water. Since 30 tablespoonfuls amount to six ounces of powder, one can calculate, as above, that the infant will receive 0·00021 ounce, equivalent to 0·0919 grain, of iron per day (equivalent to 0·54 grain of iron and ammonium citrate).

With regard to "Vi-Lactogen", an infant at the age of six weeks is given two tablespoonfuls and one and a half teaspoonfuls of powder together with eight tablespoonfuls and two teaspoonfuls of water at each feed, and is given

six feeds per day. This means 14 tablespoonfuls and one teaspoonful, or a weight of 3·5625 ounces of powder; and since "Vi-Lactogen" contains 35 parts per million of iron, the infant receives (3·5625 × 35 ÷ 1,000,000) ounce of iron, which equals 0·0001247 ounce, or at 437·5 grains per ounce, 0·0546 grain of iron per day (equivalent to 0·32 grain of iron and ammonium citrate).

If the infant were nine months old, and if one assumes it to be fed on "Vi-Lactogen", then each of five feeds would contain four tablespoonfuls and three teaspoonfuls of powder, or 23 tablespoonfuls and three teaspoonfuls, equivalent to 5·937 ounces of dry "Vi-Lactogen". This amount of powder will contain (5·937 × 35 ÷ 1,000,000) ounce of iron, which equals 0·0002078 ounce, or 0·0910 grain of iron per day (equivalent to 0·54 grain of iron and ammonium citrate).

It must be noted that all the figures for iron are as the element iron and not as the oxide.

It follows that an infant fed on "Lactogen" and "Vi-Lactogen" receives twice as much iron per day as a breast-fed infant, and many times more iron than an infant fed on diluted cow's milk. This addition of small quantities of iron is probably effective in preventing the development of an anæmia by constantly replacing the normal loss of iron from the body and keeping the iron reserve of the body at its usual level. The large doses of iron used therapeutically in anæmia are naturally not indicated before anæmia is existent.

Mackay[36] states that in two groups of artificially fed infants, one group receiving iron therapy and the other acting as controls, the difference in hæmoglobin level at the age of six months was about 7%, and by the age of twelve months it had increased to about 12%.

No attempt is made to discuss the relative merits of the various methods of infant feeding employed in this investigation. That would be quite outside the scope of this paper, the object of which is to determine the incidence of nutritional anæmia in the community, and to advance an explanation thereof if possible.

The extra iron content of "Lactogen" allows a small quantity of inorganic iron to be absorbed over a long period, stored and utilized as required. As the infants were, as far as one could tell, normal, there should have been no difficulty in absorption and utilization. The results shown in the investigation would appear to indicate that the extra iron has been absorbed, stored and utilized. The number of cases investigated, however, is too small for definite claims to be made.

An attempt was made to subdivide further the figures for "Lactogen" and "Vi-Lactogen" according to whether the mother followed the directions on the tin, or diluted the powder according to clinic instructions; the iron content of the latter mixture is less than that of the former. Insufficient cases in each group and variation in individual results did not permit a satisfactory comparison.

Prematurity.

Criteria for the determination of prematurity were difficult to define. An unsatisfactory attempt was made to classify as premature, infants who were stated to have been born three weeks or more before the expected time. Eighteen infants conformed to this criterion, and they had an average hæmoglobin level of 84·5% (11·8 grammes per 100 cubic centimetres), the standard deviation being 6·2% and the standard error of mean 1·4%.

It is to be observed that the standard error for the premature infants was considerably higher than that for all infants. This was due to the wide variation of results. The mean for premature infants selected by this criterion was higher than the mean for the whole series. This immediately suggests that the criterion of selection was unsatisfactory. To investigate this a further criterion was interposed. Infants were counted premature who had been born three weeks or more before full time, and whose weight at birth did not exceed five pounds. In making five pounds a criterion of prematurity, I took cognizance of a statement in "The Diseases of Infants and Children", by Griffiths and Mitchel (1938), to the effect that at the age of thirty-six weeks the fœtus weighed 4·3 pounds. Thirteen infants fulfilled these requirements. The mean hæmoglobin value was 73·46% (10·28 grammes per 100 cubic centimetres), the standard deviation was 10·2%, and the standard error of mean was 2·9%. The difference between the mean of premature infants and the whole population is 82·2% − 73·46% or 8·74% of hæmoglobin. The standard error of difference is 3·6%. The difference is thus 2·3 times the standard error. This difference is suggestive of significance, but is not absolute. (A difference three times its standard error is considered significant.)

From these results, it appears that premature infants, according to our standard, fail to reach by the age of nine months the mean hæmoglobin value for the whole series by 8·74%. However, some of these infants had been fed on "Lactogen" and "Vi-Lactogen", and had therefore received extra iron for a long period. It has been shown that the hæmoglobin values for infants fed on "Lactogen" and "Vi-Lactogen" are higher than the mean for the whole series. Does the same hold good for premature infants? The 13 premature infants that conformed to my standard of prematurity comprised five fed on "Lactogen" or "Vi-Lactogen", six breast fed, and two fed on cow's milk. Their hæmoglobin levels are set out below:

"Lactogen" and "Vi-Lactogen" 81%, 81%, 79%, 96%, 79%
Breast milk 76%, 66%, 68%, 42%, 53%, 83%
Cow's milk 72%, 80%

No definite comparison can be drawn, owing to the small number of cases in the series; but it is interesting to note that not one premature infant taking added inorganic iron had a hæmoglobin value below 79%, whereas out of six breast-fed infants only one had a hæmoglobin value above that level. Further investigation in a large series of cases is indicated.

The records of two somewhat similar premature infants, fed differently, are quoted here, not to prove that one method of feeding is better than another, but to illustrate certain points in regard to each case.

CASE 173.—J.McD., at the time of examination, was aged seven and a half months; the birth weight was two and a half pounds, and the child was born two and a half months prematurely. He was in hospital for four months after birth, and blood transfusions were given at the ages of five and seven weeks. His weight at the age of seven and a half months was thirteen pounds ten ounces, clothed. A blood examination gave the following information: the hæmoglobin value was 81%, the red blood cells numbered 4,980,000 per cubic millimetre, the colour index was 0·9, and the corpuscular volume was 24%. The hæmoglobin value of the mother was 76%. The infant had been breast fed for one month and was then fed on "Vi-Lactogen" to the age of five and a half months and on "Lactogen" to the time of examination.

The high iron percentage in this infant's blood may be due to one or more of the following factors: (i) Blood transfusions given in the first few weeks of life. There is no evidence that these could have other than a temporary effect. (ii) Rate of growth of the infant. Mackay[35] has shown a correlation between rate of growth and hæmoglobin level—the more rapid the rate of growth, the lower the level of hæmoglobin. This infant has certainly increased its weight satisfactorily, but not so spectacularly as the child in Case 44. (iii) Extra iron received in the "Vi-Lactogen" from the age of one month onward. Mackay[35] considers that extra iron received in small quantities is of great value when taken over a long period. (iv) Four months' stay in hospital. Josepha[18] has proved that freedom from exposure to infection prevents the onset of nutritional anæmia in infants.

CASE 44.—R.P., at the time of the first examination, was aged nine and a half months. The birth weight was two and a half pounds, and the infant was born two months prematurely. The weight at examination was seventeen and a half pounds, clothed. A blood examination gave the following information: the hæmoglobin value was 42%, the red blood cells numbered 4,850,000 per cubic millimetre, the colour index was 0·4, and the corpuscular volume was 19%. The hæmoglobin value of the mother was 91%. The infant was breast fed until the age of six months, when cow's milk, orange juice and clinic emulsion were substituted. The sister in charge of the clinic was advised to refer this patient to an honorary pædiatrician for the treatment of the anæmia. After receiving five grains of iron and

ammonium citrate three times a day for three months the child was discharged from hospital. No blood examination was made at any time during treatment. One month later I examined his blood again, with the following results: the hæmoglobin value was 70%, the red blood cells numbered 4,420,000 per cubic millimetre, the colour index was 0·8, and the corpuscular volume was 25%. The child's weight was twenty-two pounds four ounces.

This case illustrates several interesting points: (i) The rapid response of this infant to iron therapy, showing that a deficiency existed and could be easily made good. (ii) The excellent increase in weight whilst the child was under treatment, almost five pounds in four months. (iii) The necessity for all doctors and clinic sisters to realize that premature infants and infants of low birth weight frequently develop a severe grade of nutritional anæmia. (iv) The need for repeated hæmoglobin estimations whilst the child is under treatment and the necessity for continuing the therapy for many months after a normal level has been obtained. Mackay[28] considers that premature infants should have iron from the first few weeks of life until they have passed the age of twelve months, and probably even for a longer period. The infant in Case 44 was still deficient in iron after three months' therapy. It is not too much to postulate that the level would have fallen even lower without further therapy. (v) The good health of this infant with a hæmoglobin value of 42%. Kato and Emery[20] state that there must be some connexion between the low hæmoglobin content of the blood and the degree of resistance against invading micro-organisms. This infant does not illustrate that point.

Parsons and Hawksley[29] discuss very fully the blood picture in prematurity. They state that at the age of eight to ten weeks the hæmoglobin level is 45% to 55% (100% = 13·8 grammes of hæmoglobin per 100 cubic centimetres), rising gradually after that until the age of six to eight months it has reached that of the normal child (presumably the 70% to 75% found by Mackay[28]). They consider that the fall of hæmoglobin from birth to the age of three months is physiological and cannot be prevented by the administration of iron. If the store of iron is sufficient in the liver and spleen, nutritional anæmia will not supervene in later infancy. If, however, the store is not sufficient, nutritional anæmia will supervene.

Hugounenq (1899)[15] showed that two-thirds of the iron present in the body at full term was laid down in the last three months of intrauterine life. Gladstone, of New York,[12] disagrees with these findings, and states that the iron is deposited in the liver and spleen after birth and depends on post-natal destruction of blood. Parsons and Hawksley[29] state that Gladstone's arguments do not appear to furnish a satisfactory explanation of the frequent occurrence of nutritional anæmia in the premature infant or in twins.

Jennings, in New Zealand,[17] examined infants premature by from three to eleven weeks, and twins. None of his 20 subjects was found to be deficient in hæmoglobin even though some mothers were suffering from severe degrees of anæmia. The average hæmoglobin value obtained by him for children at the age of six months was almost the same as for full-time infants (namely, 12 grammes per 100 cubic centimetres). It will be noticed that Jennings's criterion of prematurity was not nearly so rigid as the one used in this investigation. It corresponded to my first attempt, and the results were similar.

Castle and Minot[3] state that prematurely born infants, twins, or young children who have peculiarities of eating are especially liable to hypochromic anæmia. They noted that the general nutrition of these infants was not often seriously affected.

Mackay[28] shows that almost all premature infants and most twins become anæmic, sometimes profoundly so. Those infants whose birth weight was about five pounds or under had a hæmoglobin value of 63·2% (five cases) at the age of eight or nine months. Kunckel[22] found an early fall in hæmoglobin value in these infants to the age of four months (46% in 21 premature infants with an average birth weight of four pounds). He stated that there was a slow, spontaneous cure in the second six months of life, but gave no figures.

Lichtenstein[24] stated that the anæmia of premature infants in the second six months of life was chlorotic, owing to the failure of the iron deposits. Josephs[19] does not consider that the low hæmoglobin value is in itself harmful, although it is probably indicative of a condition which renders the infant more likely to succumb to infection. Merritt and Davidson[32] showed that the incidence of infections was 3·6 times as frequent in untreated premature infants as in normal infants. They found an average hæmoglobin value of 10 grammes per 100 cubic centimetres (71% by my standard) among children at the age of nine months in their series of untreated premature infants. Their criteria of prematurity at birth were a length of 45 centimetres or less and a weight of 2,270 grammes. In their series of premature infants treated with iron soon after birth, the average hæmoglobin value was 13 grammes per 100 cubic centimetres (93% by my standard). They conclude that a more or less severe anæmia develops in the majority of premature infants, and that iron and ammonium citrate has proved to be a satisfactory preparation in the amelioration of the anæmia. Abt and Nagel,[1] however, doubt the efficacy of iron therapy. Hutchison[16] suggests that iron stored in the liver is unavailable for hæmoglobin formation, and only iron which overflows from the liver into the circulation can be utilized for hæmoglobin formation. Massive doses are necessary to effect this. He could find no evidence that variations in gastric acidity influenced the retention of iron, and considered that the necessity for giving large doses of iron by mouth did not depend on the poor absorption from the intestine.

Twins in Relation to Nutritional Anæmia.

Neale and Hawksley[26] describe six sets of twins with severe grades of nutritional anæmia, with hæmoglobin values varying from 18% to 50% at the time of examination. No iron or food containing iron had been given. In their cases there was a rapid response to iron therapy (five grains of iron and ammonium citrate, in some cases supplemented by ten grains of "Yestamin" given three times a day). They emphasize the value of an adequate storage of iron *et cetera* at birth in preventing the development of nutritional anæmia in twins. Most authorities agree that nutritional anæmia is as likely to occur in twins as in premature infants.

In the present investigation there were five sets of twins. They had an average hæmoglobin value of 78·3% (10·96 grammes per 100 cubic centimetres) and a red cell count of 4,540,000 per cubic millimetre.

CASES 143 AND 144.—J.D. and H.D. were born three weeks prematurely. At the time of examination their age was eight and three-quarter months. They had been fed on cow's milk from the age of two weeks. The hæmoglobin value of the mother was 86%. J.D., a male, weighed six pounds fourteen ounces at birth, and at the time of examination he weighed eighteen pounds four ounces, clothed. His hæmoglobin value was 82%, and the red blood cells numbered 3,940,000 per cubic millimetre. The corpuscular volume was 33% and the colour index was 1·1. H.D., a female, weighed five pounds four ounces at birth, and at the time of examination she weighed seventeen pounds four ounces, clothed. The hæmoglobin value was 84% and the red blood cells numbered 4,400,000 per cubic millimetre. The corpuscular volume was 31% and the colour index 1·1.

CASES 163 AND 164.—P.S. and G.S., full-time infants, at the time of examination were aged nine and a half months. They were fed on "Lactogen" and "Vi-Lactogen" from the age of two weeks. The hæmoglobin value of the mother was 90%. P.S., a female, weighed six pounds four ounces at birth, and at the time of examination she weighed twenty-two pounds three ounces, clothed. The hæmoglobin value was 78% and the red blood cells numbered 4,590,000 per cubic millimetre. The corpuscular volume was 24·5% and the colour index 0·9. G.S., a female, weighed five pounds eight ounces at birth, and at the time of examination she weighed twenty-one pounds nine ounces, clothed. The hæmoglobin value was 78% and the red blood cells numbered 4,360,000 per cubic millimetre. The corpuscular volume was 28% and the colour index was 0·9.

CASES 220 AND 221.—D.M. and P.M. were born three weeks prematurely. At the time of examination they were aged eight and a half months. They had been fed on "Lactogen" and "Vi-Lactogen" from the age of one month. The hæmoglobin value of the mother was 92%. D.M., a male, at birth weighed five pounds and at the time of examination he weighed eighteen pounds, clothed. The hæmoglobin value was 81% and the red blood cells numbered 5,140,000 per cubic millimetre. The colour index was 0·9. P.M., a male, at birth weighed five pounds seven ounces and at the time of examination he weighed twenty pounds, clothed. The hæmoglobin value was 96% and the red blood cells numbered 4,770,000 per cubic millimetre. The colour index was 1·1.

CASES 243 AND 244.—Barry C. and Bruce C. were full-time infants, and at the time of examination they were aged eight and a quarter months. They had been fed on "Lactogen" and "Vi-Lactogen" from the age of two weeks. The hæmoglobin value of the mother was 92%. Barry C. at birth weighed eight pounds, and at the time of examination he weighed twenty-one pounds eleven ounces, clothed. The hæmoglobin value was 68% and the red blood cells numbered 5,000,000 per cubic millimetre. The corpuscular volume was 22% and the colour index was 0·7. Bruce C. at birth weighed eight pounds and at the time of examination he weighed twenty-two pounds eleven ounces, clothed. The hæmoglobin value was 82% and the red blood cells numbered 4,770,000 per cubic millimetre. The colour index was 0·9.

CASES 246 AND 247.—B.G. and A.G. were born two months prematurely. At the time of examination they were aged nine and a quarter months. They were fed on breast milk to the age of four months and then on cow's milk. The hæmoglobin value of the mother was 88%. These infants received four drops of a solution of iron and ammonium citrate in their milk three times a day up to the age of six months, and from the age of eight months to the time of examination. B.G., a male, at birth weighed four pounds four ounces and at the time of examination he weighed seventeen pounds, clothed. The hæmoglobin value was 66% and the red blood cells numbered 4,500,000 per cubic millimetre. The corpuscular volume was 22% and the colour index 0·7. A.G., a male, at birth weighed three pounds ten ounces and at the time of examination he weighed sixteen pounds, clothed. The hæmoglobin value was 68% and the red blood cells numbered 3,940,000 per cubic millimetre. The corpuscular voume was 21·5 and the colour index 0·8.

The results, with the exception of those in Case 243, are not unexpected. The infants in Cases 143, 144, 163, 164, 220, 221 and 244 follow the general trend of the results throughout the whole series. The children in Cases 246 and 247 were two months premature, as well as having had very low birth weights. The low hæmoglobin value in each case was what one would expect. These infants had been given iron almost continuously and yet had a low hæmoglobin value. These results demonstrate the need for repeated hæmoglobin estimations to determine the progress of each subject under treatment. Larger doses were indicated and would probably have raised the hæmoglobin to a much higher level. The baby in Case 243, however, had a normal birth weight, had increased its weight normally, had had added iron-containing dried milk and had been a full-time baby. The reason for the low hæmoglobin value in this case may perhaps be found in the mother's statement that the child had had "bronchitis" a short time before. This malady had been mentioned by the parents in this series on a few occasions only in reply to questioning about the infant's previous illnesses.

Infants Taking Egg Yolk Regularly for Three Months or Longer.

Six infants comprised the group taking egg yolk for three months or longer. They had been taking egg yolk each day for an average period of five and a half months, were all full-time babies, and had an average birth weight of seven pounds eight ounces, and a weight at examination of twenty pounds five ounces clothed. Five had been fed on "Lactogen" and "Vi-Lactogen" from the first few weeks of life, the other had been fed on cow's milk. These infants had an average hæmoglobin value of 87% (12·2 grammes per 100 cubic centimetres); the highest was 94% (13·2 grammes per 100 cubic centimetres) and the lowest was 75% (10·5 grammes per 100 cubic centimetres); the average red blood cell count was 4,600,000 per cubic millimetre of blood.

Although the number of cases in this group is too small to permit any definite comparison to be made, it will be noticed that the average hæmoglobin value is 4·6% above the average for the whole series, but very similar to the results for "Lactogen" and "Vi-Lactogen" feeding. It would appear, therefore, that the addition of egg yolk for a long period to the diet of infants already receiving extra iron does not raise the hæmoglobin level. Whether it should affect the hæmoglobin level in breast-fed or artificially fed infants not receiving extra iron is a question that could be investigated further. The percentage iron content of egg yolk is quoted by Mackay[35] as being 0·00760%—that is, ten to fifty times as great as breast milk or cow's milk.

With regard to nutritional anæmia, iron and ammonium citrate, besides being much less expensive, is probably more efficacious and just as easy to give to the infant as egg yolk.

Mackay[20] states that in a clear case of nutritional anæmia inorganic iron, in doses which are very large compared with the total iron content of the body, produces a better response than does the organic iron present in an ordinary mixed diet containing eggs, meat, green vegetables *et cetera*.

Red Blood Cells.

Mayer[21] gives the figure of 6,000,000 red blood cells as the average for infants aged between six and eleven months in his chart for red blood cells from birth to childhood. However, he does not state on what data this figure is based. Mugrage and Andresen[34] found an average of 4,280,000 per cubic millimetre in 18 infants. Parsons[32] states that the red blood cells continue to number between 5,000,000 and 6,000,000 per cubic millimetre during infancy.

Merritt and Davidson,[33] in a series of 20 infants, gave an average of 4,740,000 red blood cells per cubic millimetre.

Faxen,[11] in a series of cases from Gothenburg, found the red cells in the latter half of the first year to number 4,500,000 per cubic millimetre.

Munday and co-workers,[25] in a series of 190 infants, gave an average of 4,820,000 per cubic millimetre.

My results for the number of red blood cells per cubic millimetre were as follows:

Male (148 Subjects).

Mean	$4{\cdot}358 \times 10^6$
Standard deviation	$0{\cdot}468 \times 10^6$
Standard error	$0{\cdot}0545 \times 10^6$

Female (128 Subjects).

Mean	$4{\cdot}3015 \times 10^6$
Standard deviation	$0{\cdot}434 \times 10^6$
Standard error	$0{\cdot}038 \times 10^6$
Difference between male and female	$0{\cdot}0565 \times 10^6$
Standard error of difference	$0{\cdot}0545 \times 10^6$

The difference is equal to its own standard error and is therefore not significant. Thus it may be stated that in this group there is no difference in the number of red blood cells between male and female children. Therefore the average of the whole series is $4{\cdot}3318 \times 10^6$.

Technique.

The red blood cells were counted in the following manner:

A Hellige red cell pipette and a Neubauer counting chamber were used. A sample of blood was collected from the thumb as for a hæmoglobin estimation. After dilution of the blood with Hayem's solution and adequate mixing, the first portion of the pipette was discarded and a sample was placed in the counting chamber under the coverslip. Then more was discarded and a fresh sample was collected in the other counting chamber. Both these samples were counted and the average was taken, so that each result given is the average of two counts of the same specimen of blood. If the results varied from each other by more than 200,000 the sample was recounted.

Discussion.

Hamil and co-workers[14] have shown that quantitatively the hæmoglobin value and number of red blood cells are

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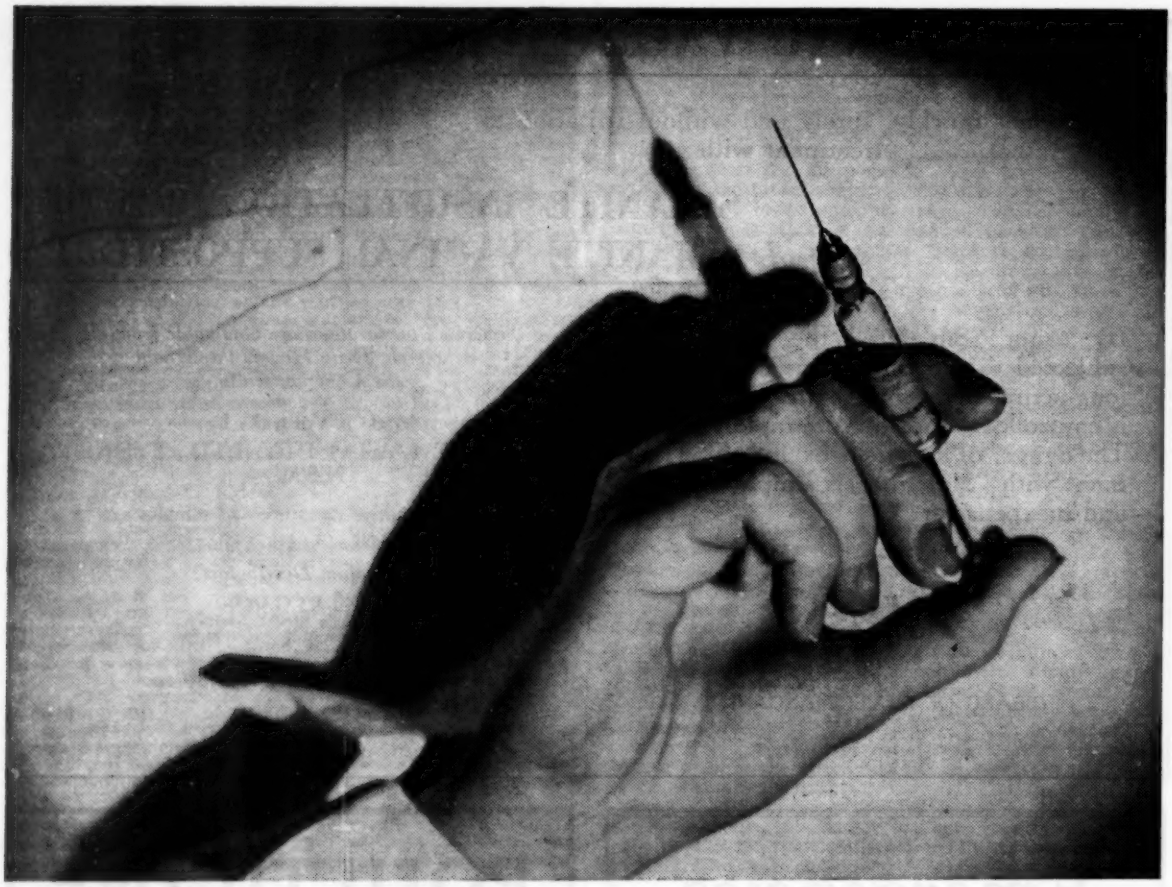

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independent of each other within certain limits, and that to know one does not enable us to estimate what the other will be. Whereas the average level of hæmoglobin of infants aged nine months in this series is above that found in Great Britain and below that found in America and Canada, the average number of red blood cells is lower than any reported from either country. Witts[50] states that the maturation of the red blood cell depends on the presence of an adequate amount of iron, copper, vitamin *C* and thyroxin, and that failure of this maturation results in the development of a microcytic anæmia. As far as could be found clinically and from the history of the patients, iron, vitamin *C* and thyroxin had been present in adequate quantities. Whether the explanation of the comparatively low figures obtained is connected with the copper intake of the infant is a matter that needs investigation. Usher and co-workers[46] have shown that there is a slightly higher level of hæmoglobin in the blood of infants taking copper in addition to iron than in that of infants taking iron alone. The figures for the red blood cell count in each group would have been of interest. The observations of Usher and his co-workers in regard to mortality rate as described earlier are of great importance.

Several infants in my series have died since the time of examination. It is intended that a review of the whole series will be made at some future date, together with an attempt to correlate the mortality and morbidity rates with various data that have been assembled. In the meantime, the figure obtained in this investigation, namely, 4,330,000 red blood cells per cubic millimetre of blood, is put forward as the average found among 276 apparently healthy infants in Sydney.

The Red Blood Cell Count and Prematurity.

The thirteen infants in this series who conformed to my criteria of prematurity, as set out above, had on an average 4,260,000 red cells per cubic millimetre of blood. The mean of this group was lower than that of the whole series, but not significantly so. No result from other countries could be found for the purpose of comparison.

The red blood count in relation to the type of feeding is shown in Table II:

TABLE II.

Type of Feeding.	Number of Children.	Mean Erythrocyte Count.	Standard Error.
Breast milk.			
Up to the age of four to six months	17	$4{\cdot}26\times10^6$	$0{\cdot}09\times10^6$
Up to the age of seven to nine months	138	$4{\cdot}11\times10^6$	$0{\cdot}037\times10^6$
Cow's milk.			
From one month onward	9	$4{\cdot}43\times10^6$	$0{\cdot}12\times10^6$
From two months onward	11	$4{\cdot}2\times10^6$	$0{\cdot}085\times10^6$
From three months onward	19	$4{\cdot}19\times10^6$	$0{\cdot}094\times10^6$
From four to six months	19	$4{\cdot}2\times10^6$	$0{\cdot}083\times10^6$
"Lactogen" and "Vi-Lactogen".			
One month onward	20	$4{\cdot}53\times10^6$	$0{\cdot}041\times10^6$
Two months onward	5	$4{\cdot}9\times10^6$	$0{\cdot}018\times10^6$
Three months onward	12	$4{\cdot}49\times10^6$	$0{\cdot}013\times10^6$
Between four and six months	6	$4{\cdot}28\times10^6$	$0{\cdot}018\times10^6$

The figures for babies fed on breast milk and those fed on cow's milk are very similar, whilst those for infants fed on "Lactogen" and "Vi-Lactogen" are slightly higher. No claim, however, can be made that blood counts on infants fed on "Lactogen" and "Vi-Lactogen" give higher figures than those made on other infants. The differences are not constant and are not significant in all groups.

However, the 138 babies, who were wholly breast fed up to the age of seven to nine months, had an average of 4,110,000 red cells per cubic millimetre of blood, whilst the 37 babies who began to be fed on "Lactogen" or "Vi-Lactogen" before the age of three months had an average of 4,570,000 red cells per cubic millimetre. No attempt is made to draw a comparison between the merits of the various methods of infant feeding, since so many factors quite outside the scope of this paper, apart from the blood picture, enter into such a comparison.

According to Faxen's standard,[11] the blood figures for the 138 breast-fed babies are below the expected normal, whilst those for the 37 babies fed on "Lactogen" and "Vi-Lactogen" were slightly above the normal. What is the factor present in these dried foods and absent from breast milk and cow's milk? The rapid response of infants to inorganic iron therapy[6][9][36][33][45] would suggest that the difference may be due to the addition of a small quantity of inorganic iron to the dried milk preparation. If a series of infants was given inorganic iron for a period of six to nine months, commencing at two months of age, with adequate controls, then valuable information might be forthcoming. It may be that small prophylactic and not therapeutic doses are all that would be necessary. Minute doses are easily tolerated over a long period, whereas therapeutic doses frequently are not.

Colour Index.

The estimation of the colour index gave the following results.

Male (148 Subjects).

Mean	0·954
Standard deviation	0·109
Standard error	0·089

Female (128 Subjects).

Mean	0·945
Standard deviation	0·093
Standard error	0·008

The difference between male and female (0·009) is not greater than its own probable error, and is therefore not significant.

Corpuscular Volume, Mean Corpuscular Volume and Mean Corpuscular Hæmoglobin Concentration.

An estimation of the corpuscular volume, the mean corpuscular volume and the mean corpuscular hæmoglobin concentration was carried out. Samples of blood from 163 subjects were tested. One prick of the thumb for each infant had to suffice to collect all samples for testing, because it was found that a second pricking invariably made the infant cry. This tended to upset the mother and I was afraid that she would tell her friends not to attend for testing. The first prick of the thumb made no infant cry after a certain technique had been developed. The corpuscular volume figures for the infants tested were as follows:

Male (83 Subjects).

Mean	26·876
Standard deviation	3·5
Standard error	0·384

Female (80 Subjects).

Mean	26·615
Standard deviation	3·6
Standard error	0·404
Difference between male and female	0·261
Standard error of difference	0·55

The difference is not greater than its own probable error and therefore is not significant.

Mean Corpuscular Volume:

Male, 83 subjects — Mean = 62·5 cubic μ.
Female, 80 subjects — Mean = 61·9 cubic μ.

Mean Corpuscular Hæmoglobin Concentration:

Male, 83 subjects — Mean = 43·1%
Female, 80 subjects — Mean = 42·8%

Technique.—The estimations were made in the following manner. A hæmatocrit with a bulb at the end for 3·8% sodium citrate solution was used. Rubber bands were employed to prevent loss of blood from the hæmatocrit during transit from clinic to laboratory. The hæmatocrit was filled with blood up to the 10 mark from a prick of the infant's thumb, and 3·8% sodium citrate solution was added to carry the blood into the bulb and prevent clotting. Rubber bands were applied to prevent leakage. The hæmatocrits were placed in a centrifuge for 45 minutes at a speed of 1,500 revolutions per minute. The result was

then read off from the hæmatocrit as a percentage of the blood collected, the bands not being removed.

Discussion.

No comparative records of the corpuscular volume, mean corpuscular volume or mean corpuscular hæmoglobin concentration in infants could be found. I am not entirely happy about the results obtained in my investigation. The rubber bands used had a tendency to slip whilst being carried from clinic to laboratory and also in the centrifuge. Many samples had to be discarded for this reason. A hæmatocrit holder made of metal also proved unsatisfactory for the same reason.

The results obtained in this series may be of use to serve as a contrast to or as a comparison with figures that will be obtained by Australian workers in the future. The corpuscular volume in relation to the type of feeding is shown in Table III:

TABLE III.

Type of Feeding.	Number of Children.	Mean.
Breast milk.		
Up to the age of four to six months ..	11	26·1
Up to the age of seven to nine months ..	84	26·28
Cow's milk.		
From one month onward	5	27·0
From two months onward	8	25·75
From three months onward	11	27·8
From four to six months	9	25·35
"Lactogen" and "Vi-Lactogen".		
From one month onward	12	26·17
From two months onward	4	27·0
From three months onward	8	27·2
From four to six months	3	27·0

The numbers in each group were too small for statistical treatment. No comment can be made about the corpuscular volume in relation to the type of feeding, owing to the general nature of the means.

SUMMARY OF RESULTS.

Two hundred and seventy-seven apparently healthy infants from the suburbs of Sydney were examined. The average age of their mothers was 28 years, and the average hæmoglobin content of the mother's blood was 83·3% (11·7 grammes per 100 cubic centimetres). The average birth weight of the infants was seven pounds six ounces and the weight at the time of examination, unclothed, was 19·78 pounds for the males and 18·94 pounds for the females. The average age at the time of examination was nine months. These infants had an average hæmoglobin level of 82·2% (11·5 grammes per 100 cubic centimetres), an average number of red blood cells of 4,330,000 per cubic millimetre, an average colour index of slightly under 1·0, and a corpuscular volume of 26·75.

Thirteen infants who fulfilled my criteria for prematurity had an average hæmoglobin level of 73·46% (10·28 grammes per 100 cubic centimetres) and an average number of red blood cells of 4,220,000 per cubic millimetre.

Twenty-one infants had a hæmoglobin level of less than 72%. An explanation for the anæmia has been advanced in 11 cases, and a possible reason has been postulated in the other 10 cases.

Five sets of twins were seen in the series. They had an average hæmoglobin level of 78·3% (10·96 grammes per 100 cubic centimetres) and an average number of red blood cells of 4,540,000 per cubic millimetre.

Infants fed on "Lactogen" and "Vi-Lactogen" had an average hæmoglobin level higher than the mean for the series by 3·6% to 6·6%, and also a slightly higher number of red blood cells per cubic millimetre. Of the 13 premature infants examined, those fed on "Lactogen" and "Vi-Lactogen" had a higher hæmoglobin level than those fed on breast milk or cow's milk. The infants taking egg yolk as well as dried milk showed no advantage over those taking dried milk alone.

There was only a weak correlation between hæmoglobin value and the following factors: morbidity of the infant, weight of the infant, parents' wages, age of mother, muscle tone of the infant. As several deaths have occurred since the completion of the investigation, a review at a later date may give further information.

CONCLUSIONS.

1. Of the series of infants investigated, 7·6% were suffering from nutritional anæmia. This percentage would have been much higher if American standards of normality had been accepted. The average hæmoglobin level for the whole series was below the expected normal of both British and American workers for infants receiving an adequate amount of iron in the diet.

2. Infants fed artificially with "Lactogen" and "Vi-Lactogen" were less anæmic than infants fed with breast milk or cow's milk. These infants also had a slightly higher red blood cell count. This was probably due to the iron and ammonium citrate added to the dried milk.

3. The blood of infants who fulfilled my criteria for prematurity had a much lower hæmoglobin level than the mean for the series. This was less pronounced in those cases in which extra iron was given in the food than in those in which it was not.

4. The blood of twins, in this series, had a hæmoglobin level only 3·9% below the average for the whole series.

5. The average hæmoglobin value of the mothers in this series was lower than the average for nurses at the Royal Prince Alfred Hospital.

6. No evidence was found in this series that the hæmoglobin content of the mother's blood influenced the hæmoglobin level of the infant, except in one case.

7. No correlation was obtained in this series between the hæmoglobin level of the infant's blood and the morbidity of the infant.

RECOMMENDATIONS.

1. Medical practitioners and nurses should be made aware of the incidence of nutritional anæmia in the community. One infant in every thirteen in this series was anæmic. The appearance of the child was of no assistance in the detection of the anæmia.

2. Clinic sisters should be allowed to prescribe iron for the infant. British and American investigators have stressed the value of iron to the infant and do not consider that overdosage will produce any ill effects, other than gastric disturbances.

3. All infants of a low birth weight or born prematurely should have iron added to their diet from an early age. Probably all infants would be benefited by the addition of iron to their food. When iron has been given therapeutically, it should be continued for a considerable time.

4. All infants should have a hæmoglobin estimation at the ages of three and nine months. A Hellige hæmometer is simple in operation and with a little practice can be used by any doctor or clinic sister. By this means the incidence of nutritional anæmia could be detected early, and the morbidity and mortality rates with which it is associated (according to many British and American investigators) would be greatly decreased.

5. A series of infants receiving all types of feeding should be given extra iron from the age of about one month, and examined over a long period of time to determine the level to which the hæmoglobin can be raised in Australia.

6. This present series should be followed up to determine the morbidity and mortality rate in relation to the hæmoglobin level. This especially applies to that group of infants comprising 3·6% of the whole series in which no reason could be advanced for the anæmia.

7. Therapeutic treatment of anæmia in infancy should always be controlled by repeated hæmoglobin estimations, and even after the completion of treatment a check should still be kept on the hæmoglobin level.

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Reviews.

A BOOK FOR MENTAL NURSES.

THERE was a time when the mentally ill were cared for by "warders" whose chief characteristic was brawn and whose function was more to guard than to cure. All this has changed and the mental nurse nowadays has to undergo a training extending over a period of three years, by which, if the necessary examinations are passed, he or she may obtain State registration as a mental nurse.

There is scope for an extension of knowledge of mental nursing to those outside the larger mental hospitals; the publication of "Psychology and Mental Disorders for Nurses", by J. W. Fisher, should aid materially in bringing this about.[1] Its great virtue lies in the fact that it contains a great deal of relevant information condensed into a small space. The book bears the same relation to the standard works on nursing as members of the "Aids" and "Synopsis" series do to more orthodox medical text-books. It is divided into two sections, the first, consisting of 35 pages, being an admirable summary of the more soundly established principles of contemporary psychology, while the second, of 78 pages, sets out briefly the signs and symptoms of the commoner psychoses and psychoneuroses, and at greater length the nursing aspects of their treatment. Free use is made of the mnemonic and of alliteration as an aid to memory; for instance, stages in *dementia paralytica* are described as: "Forty, frisky and fickle: fatty, fifty and fatuous: filthy, feeble and final"—which the medical student may find worth remembering. As a possible criticism it might be said that at times comprehensiveness has been sacrificed for brevity; for instance, the nurse is not likely to gain a very good understanding of paraphrenia from the description given; but this is inseparable from a work of this kind.

This book is confidently recommended to those concerned in the training either of mental or of general nurses. The latter should have some knowledge, however brief, of psychology and of the nursing of psychotic and psychoneurotic patients; the difficulty is to find space in an already crowded curriculum; the brevity yet comprehensiveness of this book should help to provide a solution.

FRACTURES.

NOTWITHSTANDING the production of so many books devoted to fracture work in recent times, gratification will be felt at the publication of the "Complete Outline of Fractures, including Fractures of the Skull", by J. Grant Bonnin, as a practical reference book for student study and early post-graduate days.[2] The requirements of a student's manual have been kept well in mind, with excellent help for the general practitioner, and with some value even for the specialist.

The somewhat staccato style rarely hinders clarity of expression. The free illustrations, skiagram reproductions, diagrams and sketches enable the concentration of much material into a medium-sized book. Minor errors of infrequent occurrence, such as the transposition of the subtitles of skiagrams on page 357, will no doubt be rectified in subsequent editions. A helpful and pertinent bibliography, mainly Anglo-American, follows at the end of each chapter.

The work follows modern teaching in its delimitation of the help of the physical therapist and the increased reliance placed upon early active and vocational use. Stress is laid upon the omission of avoidable clinical tests of fracture when easy access to X-ray examination is available. The early and late complications of fractures are fully discussed, and the classification of delirium as an entity among the early complications is noted. Perhaps even greater emphasis might have been placed on the necessity for the avoidance of distal œdema during treatment and on the significance of the relationship of œdema to joint stiffness. There is substantial elaboration of plaster technique, and appropriate methods of anæsthesia are discussed; but little attention is drawn to our more recent knowledge of the extreme suitability of the intravenous use of "Pentothal" in manipulative work.

There is some recognition and analysis of function in relation to alignment, and we can hope that this aspect will be more fully covered in some future work. The growing importance of traumatic arthritis receives recognition. Principles of treatment rather than the minutiæ of technique are well stressed, and there is adequate description of essential apparatus.

The methods of treatment are not restricted to those of one school of thought, and the capacity and experience of the author are reflected in valuable judgement as to the indications for individual methods. As in many similar works, clarification would be helped by reserving the term "extension", used synonymously with traction, to denote distinction from flexion.

The surgical treatment of wounds is discussed as a preliminary to the treatment of compound fractures, and strong advocacy is made for the dry treatment of recent wounds. In regard to closed wounds, the author states "a casualty department working on the dry principle will reduce the incidence of infected lacerations by 30 to 40%" over those in which is used a watery solution in any form. Exposure of wounds to the air after several hours is recommended. In infected compound fractures stress is laid upon the early identification of the organism, often neglected, with the use of appropriate corresponding chemotherapy.

The clinical picture of Sudeck's atrophy is well done, with the observation that it is still often missed in diagnosis and may follow even minor lesions.

The discouraging view of the results of treatment of non-union of the scaphoid will not receive wide approval. The present position in relation to fracture of the neck of the femur, with its operative control and diversity of after-treatment, is well set out, with able deductions.

Disability tables are appended with suitable exercise charts. It is welcome that the book includes an outline of the organization of a fracture clinic, using as a basis the report on fractures by the committee of the British Medical Association. The segregation, continuity of treatment and after-care, and unity of control have been shown to result in enormous advantages to the patient and to the industrial economy of the nation. Such development has also been recommended in the New South Wales Workers' Compensation Commission Annual Report, June, 1940. Perhaps a lead might be given by Australian Branch councils or other appropriate body in the appointment of a committee to inquire how far such organization might be locally applicable.

The imperfections of the work are few and the merits are great, and the book as an extremely able summary of the treatment of the whole range of common fractures reaches a high standard of excellence.

INDUSTRIAL HYGIENE.

INTO the 440 pages of his "Outlines of Industrial Medical Practice" Dr. Howard Collier, formerly reader in industrial hygiene and medicine at the University of Birmingham, has packed a wealth of detailed information on an aspect of medical practice which, with the increasing industrial development of Australia, will become of greater importance to many practitioners in connexion with workers' compensation.[1]

Originally designed as a text-book of industrial medicine, the work has been published in a modified form for the use of works doctors, examining surgeons and general practitioners, as well as for industrial managers and other non-medical people who are responsible for the health of industrial workers.

While there are two sections which will have no general appeal to Australian readers, namely, Part I, which deals with the executive duties of a works doctor, and Part V, on industrial forensic medicine (which necessarily is concerned only with English industrial legislation), the remainder of the volume is well worth perusal by general practitioners in Australia, particularly by those in industrial areas.

The sections on industrial environment and conditions of work are not limited to the consideration of physical factors only, but include discussions on such matters as wages, time and motion study, travelling and holidays, which are apt to be looked on as somewhat outside the scope of the medical practitioner. The chapter on noise has application to modern life generally, as well as to industrial life. About one-third of the volume is devoted to industrial medicine, and this section follows the orthodox arrangement; while it is necessarily compressed, it affords ready access to much information otherwise obtainable only in larger works. Such chapters as those on "the organic industrial solvents" and on the skin diseases of occupation may well prove somewhat of an eye-opener to those whose daily work does not normally bring them into contact with the results of some modern industrial processes on those engaged in them.

The volume is clearly printed and well indexed, and can be recommended as a useful addition to any medical reference library.

[1] "Psychology and Mental Disorders for Nurses", by J. W. Fisher, M.R.C.S., L.R.C.P., D.P.H., D.P.M., with a foreword by E. Casson, M.D., Ch.B., D.P.M.; 1941. London: Edward Arnold and Company. Crown 8vo, pp. 128. Price: 4s. net.

[2] "A Complete Outline of Fractures (including Fractures of the Skull), for Students and Practitioners", by J. G. Bonnin, M.B., B.S., F.R.C.S., M.R.C.O.G.; 1941. London: William Heinemann (Medical Books) Limited. Medium 8vo, pp. 519, with illustrations and diagrams. Price: 25s. net.

[1] "Outlines of Industrial Medical Practice", by H. E. Collier, M.D., Ch.B.; 1940. London: Edward Arnold and Company. Limited. Demy 8vo, pp. 446. Price: 21s net.

The Medical Journal of Australia

SATURDAY, SEPTEMBER 27, 1941.

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AUSTRALIA AND POST-WAR IMMIGRATION.

WHILE the British Commonwealth of Nations and its Allies must preserve the winning of the war as their central and constant aim, most people understand that the intention must go far beyond this—that though we bring all our resources to be spent and our energies to be burned in the fight for victory, it will profit us nothing unless we know what we want to do when it is all over and prepare to do it. Britain was unprepared for war, but no one will deny that she is now thoroughly awake, that she has put laziness behind her and that she gives evidence of a vision that did not seem possible a year or two ago. But Britain has done more than this; she has begun to lay plans for the difficult time that lies immediately beyond the achievement of victory over the enemy in the field. That this has been obvious to an outside observer is important. Dr. Thomas Parran, Surgeon-General of the United States Health Service, in addressing the American College of Physicians at Boston on April 24, 1941,[1] described what he had seen in England when he spent a month there as a member of a Commission on Civil Defence. He said that the whole of the population of Great Britain was in the battle line and that the whole medical profession was in the forefront of the battle. He emphasized the fact that in spite of the war, the British had not curtailed, but had extended, social insurance and other social laws and had adapted them to war needs. Moreover, British doctors in the midst of war were planning for the peace. He referred to the Medical Planning Commission of the British Medical Association, set up to "study war-time developments and their effects on the country's medical services, both present and future", and he quoted the following editorial comment from *The British Medical Journal*: "The British Medical Association now proposes to prepare for the return of peace so that medicine may be ready to meet its responsibilities in a world in which many values will be changed, fresh conceptions of society will be formed, and in which new stresses and strains will appear in the moral, material and economic fabric of the democracy we hold to be our rightful heritage." That Australian medicine is alive to the post-war problems it will have to face, will be clear to anyone who follows the pages of this journal. Apart from the purely medical problems awaiting this country, there are others in which medicine has a particular interest and one of these is immigration.

Dr. F. McCallum, Senior Medical Officer of the Commonwealth Department of Health, presented to the tenth session of the National Health and Medical Research Council a report on some medical aspects of migration. Unfortunately this valuable and timely document will not reach a large public. We should like to see a précis of it published in every newspaper in the Commonwealth. This does not imply that every citizen is or should be interested in the medical aspects of the subject; but Dr. McCallum goes into the history of migration to this country, he discusses the growth of the Australian population, he deals with the Australian attitude to migration, he considers some of the factors in failure of successful settlement, and he has something to say about administrative machinery before he deals with the future. In the years that are gone the Australian Commonwealth pursued an immigration policy and many thousands of immigrants were brought to these shores. The medical experience gained during those years will be of the greatest value in the future, for there is no doubt whatever that once this war is over many persons in the Old Country will seek new homes in this part of the world. The war has also shown to any who might have doubted it, that the nations of the earth will not always be content to remain within the confines of their own countries, and if Australia does not show a readiness to fill its empty spaces and develop its resources by opening its doors to suitable immigrants, it will have to be prepared with good reasons to justify such an attitude. The present war is being fought for freedom—freedom of the individual and freedom of the nation to which he belongs. In these circumstances it might be difficult to decide how far any nation was justified in refusing to share with others advantages of which it was not itself making full use. The whole question is one of great difficulty, and to its solution will have to be brought intelligence, courage and wisdom. It is not a question for doctors as a profession; doctors have the same interest in the problem as other members of the community, but medical knowledge can help to translate into effective action measures that are adopted by statesmen and politicians. This is what Dr. McCallum shows in his report.

Dr. McCallum starts his report by stating that migration will be one inevitable subject of post-war policies. It is, he adds, a question that cannot be left wholly within the ambit of the economist and political scientist. Though it is true that the administrative control of new settlement is a subject for research and is something in the nature of an experiment almost under laboratory conditions, there are in migration many human factors such as nostalgia, and this fact makes an experimental approach dangerous. Dr. McCallum writes: "I take it we are fighting this war to ensure a 'free and generous civilization' and that the purposes of migration are to nourish and maintain just such a 'free and generous civilization' in Australia." The

[1] *Annals of Internal Medicine*, June, 1941.

population of Australia is 7,000,000, and Dr. McCallum shows by a graph that peaks of natural increase in 1872, 1892, 1914 and 1922 have each followed the arrival of youthful immigrants in preceding years. The effects of a falling birth rate have to be considered. A rate of natural increase which attained a maximum in 1864 of 26·6‰ of mean population (with a birth rate of 42·9‰) had fallen by 1939 to 7·7‰ with a birth rate of 17·7‰. The total annual rate of increase of population had fallen to 1% in 1930, reached a minimum of 0·71% in 1935 and in 1938 was 0·98%. With our present population (in round figures) of 7,000,000 and with our present crude death rate of approximately 9·5‰ and birth rate of 17·5‰, we may anticipate 66,500 deaths and 122,500 births each year—an annual natural increase of 56,000. At the present time, on those ratios, to maintain an annual increase of 1% (or 70,000 persons) we shall require an annual quota of 14,000 immigrants. To reach an annual rate of 2% (which we maintained in the early 1920's) if there is no immediate and material increase in the birth rate, we shall require an annual quota of 84,000 immigrants. This figure was reached only at the peak of the pre-war immigration wave of 1912 when net migration figures for the year reached a total of 91,892. Having set out these figures, Dr. McCallum makes the significant remark that both natural increase and migration pay compound interest. Certain specific conditions have to be entered on the debit side of the immigration ledger. Something like 17 in every 10,000 persons have been repatriated after having become a charge upon the public. Five in every 10,000 died from tuberculosis within five years of their arrival, and an undefined proportion failed to establish themselves from a variety of factors. During the years 1929 to 1931 Dr. McCallum was Chief Medical Officer at Australia House, London, and he tried to see as many as possible of the "temperamentally unsuitable" and the "undesirable" migrants who had returned to England. They were mostly young and corresponded generally to the type of delinquent youth, the "problem case", in any environment. Among forty of these persons there were, "surprisingly" Dr. McCallum states, only fifteen who were recorded as aments. Another fifteen were behaviour psychopaths, border-line dements. Of the other ten, eight were poor stock, rolling stock, prostitutes and thieves, by nature rather than by nurture "morally irresponsible persons". The other two, man and woman, were confidence man and courtesan, with ability and personality far above the average. This group, in Dr. McCallum's opinion, represented a stratum of any society, but it is, he adds, typical of a class of migrant who lacks "the necessary capacity for adapting himself to the demands of his environment". In regard to tuberculosis he points out that in any case of tuberculosis there is an involved play of individual, family, social, economic and environmental factors, but that this complexity of factors is exaggerated when the tuberculous person is a migrant. The general conclusion is that the great bulk of the immigrants have been successfully assimilated in the Australian population. The immigrants have been "fair average quality—in the mass neither materially better nor worse than their fellow citizens of their country of origin, nor of their country of adoption". The administrative machinery of immigration functioned most satisfactorily between 1925 and 1932. It is not necessary to trace the history of this administration, but it must be pointed out that at the highest peak of its effectiveness there was a roster of medical referees in different parts of the United Kingdom and in some centres of the Continent who examined applicants before they were accepted for assisted migration. (A similar arrangement was recently described in this journal in connexion with the Canadian Public Health Service.) There was also a staff of lay selection officers. Every case marked doubtful on medical grounds was referred for review and decision to the Chief Medical Officer at Australia House, London. Since 1932 there has been no chief medical officer to do this work; his place has been taken by a part-time officer.

In regard to the future, Dr. McCallum wisely remarks that if, when immigration is resumed, we are to retain the principles of selection and restriction, we should be prepared to consider critically just what are our aims. This, as we have already pointed out, is going to be no simple business. The following is a summary of Dr. McCallum's remarks in what is probably the most important part of his report. Fitness as we know it is essentially a relative term. The capacity of immigrants to adapt themselves to a new environment must be governed largely by their possession of a "*mens sana in corpore sano*". Beyond this there is a capacity defined by the late J. S. Purdy as characteristic of the "sticker". It is, as Karl Pearson stated, essentially the man who makes his environment and not the environment which makes the man. There is yet another quality without the possession of which all the rest are of no use; this is described in "The Emigrant's Guide" as that quality which enables a man to overcome the scruples, the remonstrances and the wailings of his wife. In an investigation in Canada into the "Three Thousand Families" scheme every failure was almost entirely due to a failure of the woman to settle down. From the woman's point of view it is necessary that she should have an adequate house. Dr. McCallum assumes that when immigration is resumed the principles of the *Immigration Act* will be retained. He also assumes that any relaxation of these principles will be granted, never as political expediency, but only after careful consideration of the implications of any departure from them. Experience has shown that the present restrictions are intelligible and workable. Dr. McCallum does not see the need for any more elaborate machinery than at present exists. He thinks that possibly in the future microradiography may be demanded in the examination of all migrants, and in regard to the psychological condition of migrants he is convinced that an Australian medical man alone can make a balanced assessment of the physical and mental fitness of the migrant who is to become an Australian citizen and progenitor of future Australians. With this most Australian doctors will agree.

No discussion on immigration and Australia can be complete without reference to the White Australia policy. Dr. McCallum states that if in a post-war reconstruction we have to justify that policy in an international tribunal, there is probably no more concise statement of our acceptance of that policy than that expressed by Sir John Latham: "It is firmly and, in my opinion, reasonably believed by Australians that Australia can do most to secure what the Greeks would have called 'a good life' for her people by maintaining the present social composi-

tion of the community. This is not a selfish ideal, for it is, we believe, as a free white democracy that Australia can make her best contribution to the peace and well-being of the world as a whole." Of course the question is not always one of white and coloured people. The "alien" whites create much greater difficulties and there arises "a clash of impulses, generosities and prejudices". Into this subject we do not propose to go further, except perhaps to state that Australians generally err on the side of generosity rather than on that of prejudice—they are, all things being equal, willing to give a man what is known colloquially as a "fair go". But we have already stated that the solution of this whole problem will call for intelligence, courage and wisdom; we may now add that unless generosity in this matter is founded on knowledge and guided by judgement, it becomes foolishness and may in the final event prove to be sheer insanity. In any case we can agree with Dr. McCallum that the medical principles and aspects as discussed by him will be very relevant in administration and control of migration; the medical profession has the duty of guarding the health of immigrants as zealously as it guards the health of native-born Australians.

Current Comment.

TWO RARELY RECOGNIZED PULMONARY DISEASES.

As far back as 1905 Aldo Castellani described a condition that he called bronchomoniliasis—a disease of the lungs caused by a fungus of the genus *Monilia*. In "A Manual of Tropical Diseases", by Castellani and Chalmers, published in 1919, the disease is given a brief though clear description. Castellani has written about it in other places on a number of occasions. *Monilia* may appear as a secondary invader in other diseases of the lung. It may be that it is frequently present in the bronchi without causing any disability or producing any lesion. Perhaps for this reason and because Castellani is known to be particularly enthusiastic about mycotic disorders (some people may regard him as a crank), bronchomoniliasis has not received until recently a great deal of attention. But its existence has long been accepted, and it has been recognized by a number of observers in various parts of the world. It is the first of the two rarely diagnosed conditions to which the title of this article refers. Our attention has recently been directed specially to moniliasis by a well-informed article written by C. J. Koerth, J. M. Donaldson, junior, and R. G. McCorkle.[1] The words "rarely recognized" in relation to this disease apply to Australia, but not, apparently, to some other parts of the world. For example, as Koerth, Donaldson and McCorkle point out, Keiper concluded that 2·93% of pulmonary disease was due to *Monilia*. Bronchomoniliasis is commoner in tropical countries; but it may occur at any latitude. The pathological changes in the lungs are generally similar to those of tuberculosis. Consolidation, cavitation, fibrosis and tubercles are found. Microscopic examination reveals "thickening of the alveolar walls and tubercles composed of a caseous centre, surrounded by epithelioid cells, lymphocytes and an occasional giant cell. . . . No lesions have been described which are generally accepted as pathognomonic of bronchomoniliasis". If it is true that such extensive damage to the parenchyma of the lung can occur, the name "bronchomoniliasis" is not a very good one.

Koerth, Donaldson and McCorkle state that three clinical types of the disease have been described. In the milder type the patient's general condition is little affected; cough is slight; small quantities of mucopurulent sputum are voided; no fever occurs. Physical examination reveals either a few scattered râles or no abnormality. After several months spontaneous cure occurs, or the condition passes into the severe type. In the moderately severe type the picture is that of "bronchial catarrh with fever, cough and dyspnœa". The sputum may be blood-stained. Signs of bronchitis are revealed by clinical examination. This state may continue for years, or the severe form of the disease may supervene. "The severe type resembles pulmonary tuberculosis in its clinical manifestations and frequently in its X-ray picture." Death as a result of this form of the disease is not uncommon.

In addition to these types, tracheobronchitis, lobar pneumonia, asthma and chronic bronchitis due to infection by *Monilia* have been described.

Diagnosis depends mainly on the constant presence of *Monilia* in the sputum and the continuous absence of tubercle bacilli. The mere presence of *Monilia* is not sufficient to justify a diagnosis of moniliasis; culture and study of the fungus are necessary. Tests of virulence consist in intravenous or intrapulmonary injection in the rabbit. Agglutination tests and skin tests of sensitivity are also employed.

Koerth, Donaldson and McCorkle report two cases. In each case the patient's blood serum caused agglutination of suspensions of conidia of the suspected fungus, while serum from normal controls did not. A test of sensitivity was employed in the second case only; the patient reacted. In one case an ulcer from which *Monilia* was cultivated appeared on the pharyngeal wall. The radiological appearances were suggestive of tuberculosis excepting for the rapid changes that took place in one case. In the other case infiltration at each apex and cavitation at the right apex were noted. Iodides have been found useful in treatment. Lipiodol by intramuscular injection or intratracheal instillation has been recommended.

We would suggest that medical practitioners in Australia should be on the lookout for pulmonary moniliasis. We are rather doubtful about the importance of this disease, just as we are doubtful of the accuracy of some of the descriptions of it. It may well be that in many cases sputum culture would reveal the tubercle bacillus. On the other hand, medical men here may have been attributing to the tubercle bacillus many morbid conditions that are really due to *Monilia*. A careful study in Australia, controlled by sputum culture and animal experiment, would be welcome.

The second rarely diagnosed disease that we have in mind is a condition that has become known as lipoid pneumonia. This has been discussed recently, in relation to a case that they report, by J. J. Singer and L. J. Tragerman.[1] They draw attention first to an editorial article in *The Journal of the American Medical Association* relating to the dangers of the promiscuous use of nasal oil sprays and the internal administration of oils. "Lipoid pneumonia" is most likely to occur in infants, old people, feeble people and people suffering from certain neurological disorders. "Clinically significant lesions in otherwise fairly healthy adults have not been reported with such frequency." In the case recorded by Singer and Tragerman the patient was a medical man, who had been in the habit of taking liquid paraffin for the relief of constipation. He was subject to spasm of the glottis and dyspnœa. Swallowing of liquids was apt to cause cough, followed by this spasm of the glottis. He coughed blood-streaked sputum. Every few weeks he suffered from a febrile illness with a severe cough. After a few days of fever he would cough up thick purulent sputum, which had a foul odour. He became emaciated, weak and breathless, and he suffered from pain in the chest. A diagnosis of carcinoma of the lung had been made before the patient was seen by Singer and Tragerman, although the X-ray appearances were more suggestive of pulmonary fibrosis. In 1938 Singer and Tragerman made a diagnosis of bronchiectasis, abscesses and emphysema. Although the

[1] *The American Review of Tuberculosis*, June, 1941.

[1] *The American Review of Tuberculosis*, June, 1941.

sputum was examined on a number of occasions, the presence of oil droplets was noted once only. X-ray examination showed appearances suggestive of atelectasis at the base of each lung, as well as an extensive area of increased density in the lower zone of the right lung. The patient refused bronchoscopic examination. He died about a year later of congestive cardiac failure during a severe febrile illness. Post-mortem examination revealed "chronic lipoid pneumonia in portions of all lobes, with severe pulmonary fibrosis, bronchiectasis and emphysema; pulmonary arteriosclerosis with thrombosis; chronic *cor pulmonale* with congestive heart failure; terminal bronchopneumonia". At histological examination it was found that large portions of the parenchyma of the lower lobes were replaced by dense collagenous connective tissue; cystic spaces containing lipoid material were noted; numerous macrophage cells containing fat vacuoles were seen. On analysis the oil from the lungs was found to have the characters of a mineral oil. It is assumed that the patient frequently aspirated small quantities of liquid paraffin into his lungs because of some disturbance of the swallowing reflex. "Lipoid pneumonia" occurred and eventually led indirectly to his death.

This condition may be considered from two points of view. The first has to do with its nature. From reports that have appeared in the literature it is clear that the condition may not be characterized by pneumonia and that it may not be caused by a lipoid—liquid paraffin is a hydrocarbon and not a lipoid. The substance that is inhaled, however, be it oil or liquid paraffin, appears to give rise to a reaction in the lung cell that produces a lipoid degeneration; the end result, as in the case described, is a fibrosis. The patient whose history is described by Singer and Tragerman suffered from time to time from febrile illness and coughed up purulent sputum and may have had recurrent attacks of pneumonia. In some reports the term paraffinoma has been applied to a lesion in the lung caused by the inhalation of liquid paraffin. It would seem, however, that such a term would apply to a lesion that was more or less circumscribed. These points are mentioned to show that this subject is one in which a great deal more work must be done before finality regarding the pathogenesis can be reached. In connexion with the second point of view we should remember that the instillation of oily substances into the nasal passages and the administration of oil to infants and children *per os* are everyday procedures everywhere. It would not be surprising to learn that these practices were responsible for illness of greater or less severity, perhaps in many cases quite transient illness. At any rate, it is worth bearing in mind that ill-advised medication with oil may be responsible for extensive damage to pulmonary tissue and perhaps death.

CARCINOMA OF THE STOMACH.

SIXTY years have now passed since Billroth performed the first successful partial gastrectomy for carcinoma of the stomach in 1881. By successful is meant that the patient had an uneventful convalescence and was discharged from hospital twenty-two days after operation. While the ultimate result of death within four months from peritoneal metastases was less of a success, this does not detract from the praise due to Billroth as a pioneer in gastric surgery, especially when we remember that the present-day partial gastrectomies (which incidentally are only modifications of Billroth's two operations) are in many cases followed sooner or later by metastases and death, the comparative rarity of five-year cures of gastric carcinomata being well known. The necessity for earlier diagnosis of gastric carcinoma as the first step towards the reduction of the mortality rate of this disease has been repeatedly emphasized; and the desirability of suspecting and investigating thoroughly all adult patients who complain of dyspepsia or debility in any form whatever is being gradually instilled into the minds of medical practitioners by all authors on this subject.

The criteria necessary for the early diagnosis of gastric carcinoma and accordingly for effecting a reduction in the mortality figures, have been summarized by T. F. Mullen[1] under the headings pertaining firstly to the patient and secondly to the physician, as (i) a sensitive patient, (ii) a suspicious clinician, (iii) a studious and searching Röntgenologist and gastroscopist, and (iv) a superskilful surgeon. The concept of a sensitive patient is one which has in the past received little notice, but has an obvious and important bearing on the subject. Mullen points out that contrary to expectations the patient who is sensitive to mild degrees of digestive discomfort is not the emaciated, cachectic and vomiting person who has suffered from pain and increasing distress for from eight months to a year before seeking medical advice. Rather he is more likely to be ruddy cheeked, and probably has not previously had digestive difficulty. He is apt to be more annoyed than impressed with his symptoms, and may have difficulty in describing his complaint, being only able to state that he has a "little stomach trouble", that is, a feeling of fullness in the epigastrium, a smaller capacity for food, a tendency to eructate, a feeling of motion or of warmth in the epigastrium, and occasionally a mild degree of nausea. The sensitive patients who have had the symptoms of peptic ulcer for years have grown familiar with its pattern, and therefore will note with the onset of more serious trouble that there has occurred a change in the degree or rhythm of the symptoms. Food may now tend to increase the pain rather than to relieve it, and there persists a constant mild degree of nausea with occasional vomiting. The functional dyspeptic who for years has been uncomfortable but relatively healthy, notices the advent of new and more persistent symptoms—nausea, more or less continued discomfort, and the onset of bad health in place of the previously relatively good health. The sensitive patients who do not develop dyspepsia may notice early in the course of the disease a loss of physical and mental stamina. Such a patient suddenly tires easily, is less able to do his work or hobby, and is physically unable to carry on without great effort; he has to spur himself to do what he formerly did with zest. Having noticed these symptoms, so often mild, the sensitive patient is fortunate if he has as his medical adviser a suspicious clinician. According to Mullen, clinicians may be divided into two classes: (*a*) those who are eternally suspicious of carcinoma of the stomach, but never find it, and who are apt to complain of the futility of their efforts, and (*b*) those who are never suspicious, eternally miss the diagnosis and comment on the hopelessness of the disease. The suspicious clinician, with the aid of the studious and searching radiologist and gastroscopist, must find that his suspicions are sometimes justified. From N. Owen's figures Mullen calculates that at any one time there would be 150,000 cases of gastric carcinoma in the United States of America and that 27,000 of them would be suitable for operation that might be followed by cure. On a similar basis there would be at the moment in Australia approximately 1,000 cases of gastric carcinoma amenable to operative treatment. The more suspicious the clinician and the more studious and searching the radiologist and gastroscopist, the more of these 1,000 cases might be discovered before metastases occur.

No relationship was found between the regional lymphatic spread of carcinoma of the stomach and the duration of symptoms in a series of 53 cases studied by E. B. Kay,[2] who unfortunately does not indicate what he considered the onset of symptoms. In a study of this kind it is important to differentiate between obvious symptoms of vomiting, loss of weight, pallor, dyspnœa and pain, and the vague, indefinite, early symptoms of carcinoma of the stomach, for it is to be expected that the onset of these vague symptoms would be more closely related to the development of the malignant change. In this series Kay found no relationship between the size of the neoplasm and the presence of metastases, but, as expected, he was able to demonstrate a definite relationship between the macroscopic types of tumour and the incidence of

[1] *Archives of Internal Medicine*, April, 1940.

[1] *Surgery, Gynecology and Obstetrics*, February 15, 1941.
[2] *Annals of Surgery*, June, 1941.

lymphatic metastases. The sessile neoplasms had an incidence of metastases of 95·4%, in contrast to 60% for the polypoid neoplasms. In a radical mastectomy for carcinoma of the breast the regional lymph glands, axillary fat and the breast are all removed *en bloc* irrespective of the size of the tumour, the duration of symptoms or the presence of palpable lymph nodes; and in order to reduce the operative mortality in cases of carcinoma of the stomach, it is essential that the operative procedures on these patients should also be so designed as to include all possible zones of lymphatic metastases regardless of the duration of symptoms, the size of the neoplasm, and the presence or absence of palpable lymph nodes. From the Cleveland Clinic, G. Crile[1] is able to report a mortality rate of 7% among patients who have had gastric resections performed for carcinoma of the stomach, and he attributes this to the employment of local anæsthesia reinforced by the intravenous administration of "Sodium Pentothal" as an analgesic agent, not as an anæsthetic agent. His preference is for this type of anæsthesia because sufficient relaxation may be obtained while the depressing effect of a general anæsthetic is obviated, stimulation of respiration and the inhalation of mucus are avoided, the blood pressure is not depressed, and the patient's nervousness and discomfort may be controlled by the addition of small amounts of "Pentothal". In the past the bugbear of gastric resections has been the high incidence of post-operative pulmonary complications; but this should be partly overcome by the use of such an anæsthetic. Crile also recommends a post-operative course of sulphapyridine or sulphathiazole as an additional safeguard in reduction of the incidence of post-operative pulmonary complications. However, though there is no doubt as to the efficacy of these drugs once pneumonia has developed and their use in these circumstances will certainly lower the post-operative mortality rate from pneumonia, the wisdom of a prophylactic course in all such cases must be seriously questioned until the toxic effects of these drugs have been more clearly defined.

At the Mayo Clinic over a period of thirty-two years (1907 to 1938) the diagnosis of malignant disease of the stomach was made in regard to 10,890 patients; of this number, 6,352 underwent operations and 2,840 of these were resections. This extensive series of cases has been reviewed by E. B. Lewis and a preliminary report of the conclusions drawn from this review has been published.[2] The treatment for a tentative period with a restricted milk diet and alkalis of a person who presents symptoms similar to those caused by a peptic ulcer, to determine by his response to the treatment whether or not the lesion producing the symptoms is malignant, has been shown to be valueless, for in this series 81% of the patients had received such treatment and had responded in the manner characteristic of patients suffering from benign ulcers. Among those patients on whom gastric resections were performed, Lewis was able to demonstrate a definite relationship between the degree of gastric acidity and the mortality rate. He divides the patients into three groups: (*a*) those who had achlorhydria, (*b*) those who had a free acidity of less than 30 units, and (*c*) those who had free acidity of 30 units or more (Töpfer's method). For patients in the first group the mortality rate was 20%; for those in the second, 11%; and for those in the third group, 10%. Many years ago an observation was made to the effect that the decrease in gastric acidity of patients who have gastric malignant lesions is a general index of the severity of the disease; but this aspect has received very little attention. From his series of cases Lewis makes the paradoxical observation that patients who had a short duration of symptoms had lesions more malignant than the lesions of those whose symptoms endured longer. He reaches this conclusion on account of the smaller proportion that could be treated by operation with hope of success. A similar observation was made when the duration of symptoms was studied in relationship to survival. For those patients whose symptoms had been present for less than a year the five-year survival rate was 25%, whereas for patients whose symptoms endured for more than a year the five-year survival rate was 32%. It has already been mentioned that Kay was unable to find any relationship between the regional lymphatic spread of carcinoma of the stomach and the duration of symptoms in his cases. Lewis suggests that his paradoxical observation is probably due to the fact that those patients whose symptoms have endured for more than a year represent a more highly selected group of individuals than is usual; the fact that they have lived so long might, he thinks, be expected to indicate that the patients were more amenable to treatment in spite of the presumably long course of the disease with which they are afflicted. Although it was of necessity time-consuming, the importance of an investigation such as that of Lewis is apparent; it has the stamp of high quality that we have learned to expect from the great wealth of clinical material available at the Mayo Clinic.

[1] *Cleveland Clinic Quarterly*, January, 1941.
[2] *Proceedings of the Staff Meetings of the Mayo Clinic*, October 2, 1940.

"THE MEDICAL ANNUAL."

THE publication of "The Medical Annual" for 1941[1] is an event on which the medical profession can and should congratulate the publishers. Readers are informed in a publisher's note, appearing immediately before the introduction, that this volume, the fifty-ninth of the series, has been produced "in circumstances which have presented serious difficulties, not only to the publishers, but especially to the Editors and Contributors". The medical profession may regard itself as particularly fortunate to be served so well in times of national stress. Australian practitioners who manage to secure a copy of this most valuable work will be the envy of their fellows, for we understand that many copies destined for this country have been lost or destroyed by enemy action.

In this year's issue the number of references to foreign literature is smaller than usual, since few European journals have been available. This does not appear to have affected the book adversely. It is true that articles, for example, on war surgery have not been very numerous, but we are told that this deficiency will be made good in the next volume—many recently published articles appeared too late for inclusion in the present volume. For those who do not know this work, it should be stated that it covers the whole of medicine. The subjects are arranged in alphabetical order and each has been reviewed by a contributor known to be eminent in the sphere to which the particular subject belongs. Occasionally an editorial comment is added, and after every discussion a list of references to journals is given. As is usual, many of the sections are of outstanding importance. References are made to sulphanilamide compounds and their uses; the more important of these deal with their exhibition in the treatment of infants and children, of dermatitis and of war wounds. These drugs are also mentioned in many other sections and in connexion with many pathological conditions, such as Ludwig's angina, gonorrhœa, scarlet fever, trachoma and phthisis. It is important to remember that much of the work reported in connexion with sulphanilamides is almost experimental, and that reliable deductions cannot be drawn from results obtained in a handful of cases. The section on the thyreoid is full of useful information, as is that on hormone excretion. Practitioners with an interest in law will find the section on legal decisions and legislation attractive. Gastric and duodenal ulcer are considered extensively from both the medical and surgical aspects; the sections on melena and on perforation are useful and the section on treatment shows that careful study is still necessary in this difficult subject.

The subjects mentioned are merely examples of many others that might be chosen. It is impossible to review a book like this; that is not our intention. What is intended is to let practitioners know that it has been published once more and to welcome its appearance.

[1] "The Medical Annual: A Year Book of Treatment and Practitioner's Index", edited by H. L. Tidy, M.A., M.D., and A. R. Short, M.D., B.S., B.Sc., F.R.C.S. Bristol: John Wright and Sons Limited. Demy 8vo, pp. 561, with illustrations. Price: 20s. net.

British Medical Association News.

SCIENTIFIC.

A MEETING of the New South Wales Branch of the British Medical Association was held on May 22, 1941, at the Royal Prince Alfred Hospital. The meeting took the form of a number of clinical demonstrations by members of the honorary staff of the hospital. Part of this report appeared in the issue of September 13, 1941.

Darier's Disease.

DR. CLIVE F. ROBINSON showed a female patient, aged sixty-five years, suffering from Darier's disease. She said that she had had an eruption for thirty years. It was present on the scalp, on the face, just below the nape of the neck, over the *manubrium sterni*, on the lower part of the back near the coccyx and in the anal and genital regions. The lesions consisted of individual papules, which in most places had coalesced, situated at the mouth of pilo-sebaceous follicles. In the anal and genital region the lesions had become vegetative. Dr. Robinson said that it was proposed to give the patient large doses of vitamin *A*, treatment with "Solganal B", and irradiation with X rays. The prognosis as to cure was bad.

Pityriasis Rosea.

Dr. Robinson next showed a girl, aged eighteen years, suffering from *pityriasis rosea*. The condition had begun with what she described as an oval-shaped "ringworm" on her loin; now oval, elongated, yellowish-pink macules were distributed in a roughly symmetrical fashion on the body and neck. The condition was not irritable. As the disease was self-limited, no treatment was required.

Secondary Syphilis.

Dr. Robinson's next patient was a woman, aged fifty-three years. Dr. Robinson said that he had been told that she had been "put off" the dole and told to earn her own living. Small and large multiple papules, reddish and coppery brown in colour, were symmetrically distributed over her face and body. Glandular involvement was present. Dr. Robinson proposed to give her during the first year of treatment 12 grammes of "Novarsenobillion" and 10 grammes of bismuth; after that the case would be reviewed.

Psoriasis.

Dr. Robinson then showed two patients suffering from psoriasis. The first was aged thirty years and the second forty-five years. The first had an unusual distribution of plaques on the lower third of both legs; the extensor surfaces of the knees and elbows were not involved, although the same papules were beginning to appear on the forearms. The second patient was a stout woman with huge plaques, eight to ten inches in diameter. The plaques were typical of psoriasis; they were coalesced papules covered with shiny silvery scales. Dr. Robinson said that he proposed to treat both patients by the administration of arsenic internally, the use of coal tar and "Vaseline" ointment externally, and the exposure of the patient to ultra-violet light.

Erythema Multiforme.

Dr. Robinson finally showed a man, aged fifty years, who had acute, inflammatory, dark red papular lesions, tending in places to become papulo-vesicular, on the dorsum of both hands. The lesions had appeared overnight. Dr. Robinson said that he proposed to treat the patient by the application of calamine lotion and the removal of his unhealthy tonsils.

Intracerebral Hæmorrhage (Right Frontal Lobe).

DR. W. LISTER REID showed a male patient who had suffered from severe headache and soreness at the back of the eyes for three months, and from epileptiform seizures involving the left side of the face.

Examination revealed slight weakness of the left side of the face and the left upper extremity, some mental dulness and disorientation, bilateral papilloedema, and some incoordination of the left upper extremity. Lumbar puncture was performed and the cerebro-spinal fluid was found to be under a pressure of 310 millimetres; the fluid contained 200 milligrammes *per centum* of protein. The Wassermann test failed to produce a reaction.

A right fronto-parietal osteoplastic craniotomy was performed. When the dura was opened the lesion was found framed in the opening. It was fairly discrete, and the surface was mottled with variegated colours, as in old hæmorrhage. The mass was firm, and it was possible to dissect it out *en bloc*. At the completion of the operation the patient was fully conscious and felt perfectly well. He made an uninterrupted recovery.

Hodgkin's Disease.

DR. HAROLD HAM showed a young woman, aged twenty-one years, who had first been referred to him on June 4, 1939, with a history of a lump on the right side of the neck, present for six months, and a lump over the sternum, present for five months. There was no history of cough or dyspnœa. On examination at that time some bulging of the sternum and ribs in the region of the second right sterno-costal articulation was noticed. There were three fairly large, rubbery, discrete glands in the right supra-clavicular region and posterior triangle, and two enlarged glands in the left axilla. The spleen and liver were impalpable.

X-ray examination of the chest revealed quite extensive enlargement of the mediastinal glands on the right side at the level of the arch of the aorta; this was considered to be due to either lymphadenoma or lymphosarcoma.

The blood count revealed no definite abnormality, except slight leucocytosis. No eosinophilia was present. The blood failed to respond to the Wassermann test or to the Kline precipitation test.

A small gland in the right posterior triangle was excised for biopsy, and the pathologist reported that the appearances suggested a diagnosis of Hodgkin's disease.

Dr. Ham said that deep X-ray therapy was given in moderate doses, first of all to the right side of the neck, later to the mediastinum and left axilla. Treatment was completed on September 28, 1939, and no further treatment had been given since then. The patient had remained apparently well; the enlarged glands had disappeared, and X-ray examination of the chest one month after treatment revealed much diminution of the mediastinal mass. The patient had reported at intervals for X-ray examination, and the latest one, made just prior to the meeting, revealed no enlargement of the mediastinal glands. Dr. Ham pointed out that this patient had been kept in good health for nearly two years, even though there had been involvement of the mediastinal glands. It appeared possible that she would continue in good health.

Carcinoma of the Mouth.

Dr. Ham also showed five patients who had had carcinoma of the mouth treated by irradiation. The first three had had quite extensive epitheliomata of the lower alveolus, in two cases extending well onto the floor of the mouth. One patient, who had first been seen in November, 1937, had been treated by deep X-ray therapy, followed by the application of a radium dental mould. The patient had remained perfectly well for three and a half years. The other two patients with carcinoma of the lower alveolus had been treated by the application of a radium dental mould only, and appeared well some eight months and six months respectively after treatment. Prophylactic dissection of the cervical glands had not been carried out in these three cases, as it had been considered quite justifiable in view of the site of the lesions to watch the patients carefully at intervals of one month.

Dr. Ham's next patient was a man, aged seventy-five years, who had attended the Dental Hospital in April, 1940, because his upper denture was troubling him. A biopsy was taken and the pathologist reported squamous carcinoma. He was admitted to the Royal Prince Alfred Hospital two months later, and the honorary surgeon considered that the condition was a carcinoma of the palate of a low grade of malignancy, and that surgical interference was contra-indicated. He was therefore sent for irradiation therapy and was first seen by Dr. Ham on September 25, 1940. On examination at that time a large, shallow, epitheliomatous ulcer was present at about the centre of the hard palate, with a further small lesion involving the left upper alveolus. The mucosa over most of the rest of the hard palate gave evidence of possible early malignant change. There were no palpable glands in the neck. X-ray examination of the maxilla revealed no evidence of bone invasion. The blood failed to respond to the Wassermann test.

Deep X-ray therapy was given from October 8 to 31, 1940, two fields being used, one directed obliquely lateral to the maxilla on each side. The voltage used was 375 kilovolts and the dose to each field was 3,200 *r*.

Dr. Ham said that the interesting fact in this case was that a slow-growing adult type of squamous carcinoma had proved to be highly radio-sensitive. This was not so uncommon as some writers led one to believe.

At the time of the meeting the patient gave no evidence of the lesion, the mucosa being smooth and clear. There was no evidence of involvement of the cervical glands.

Dr. Ham's last patient was a man, aged sixty-one years, whom he had first seen in February, 1939. On examination at that time an epitheliomatous ulcer was found, involving the left tonsil and extending along the lower edge of the soft palate and the posterior pharyngeal wall to the uvula, which was completely involved. There were no palpable cervical glands. The patient was first given an intensive course of deep X-ray therapy to each side of the neck. This produced an appreciable reduction in the lesion. Later, early in July, 1939, an implantation of gold radon seeds was carried out. Twelve seeds, each containing 1·1 millicuries, were implanted over and around the area of the growth. The patient had remained apparently well since. He had been referred back to the surgeon; but no dissection of the cervical glands had been undertaken, as he was not a good "operative risk".

Medical Societies.

MELBOURNE PÆDIATRIC SOCIETY.

A MEETING of the Melbourne Pædiatric Society was held on July 9, 1941, at the Children's Hospital, Melbourne, DR. H. L. STOKES, the President, in the chair. The meeting took the form of a series of clinical demonstrations by some of the members.

Bilateral Congenital Dislocation of the Hip.

DR. H. DOUGLAS STEPHENS showed a male child, aged three years, with bilateral congenital dislocation of the hips. There was an unusual association with deformity of the knee of the *genu valgum* type. The femora were short, the acetabula were extensively shallow and the epiphyses for the femoral heads had not yet appeared. No treatment had as yet been instituted, and Dr. Stephens sought advice in this direction. He preferred not to offer a prognosis.

DR. E. PRICE agreed that the case presented unusual features in the complete absence of acetabula, the undeveloped upper ends of the femora and the poor differentiation into the neck and trochanter.

Dr. Price thought that the condition might even fall into the class known as phocomelus. Further X-ray films of both femora and comparison of their lengths would be instructive. The knee deformity had something in common with the well-known familial displacement of the patella. Bilateral congenital dislocation of the hips in boys in his clinic had been attended by uniformly unsatisfactory results. Dr. Price did not think that Dr. Stephens could stabilize the hips in the acetabula. By traction it might be possible to bring the right hip down and some form of shelf-operation might be successful.

DR. K. H. HALLAM was interested in the ultimate appearance of the epiphysis when the function of the muscles moving the hip joint was in abeyance. Dr. Hallam said that the degree of dislocation present was extreme and the resulting muscular atrophy and loss of power led to a considerable reduction in the stimulus to bony growth.

Congenital Talipes.

DR. W. KENT HUGHES spoke on congenital talipes. He said that 75% of patients could be cured with manipulation and retentive apparatus; 20% needed tendon operation. The remainder required bone operation for a perfect result. Dr. Kent Hughes showed a film demonstrating his operation of wedge tarsectomy.

Cerebellar Ataxia.

DR. ROBERT SOUTHBY showed a boy, aged six years and three months, suffering from gross ataxia. The parents were normal, healthy people, and he was the only child. The mother was quite well throughout the pregnancy and had had an easy non-instrumental delivery, labour lasting six hours. The child's weight at birth was five pounds, and it had increased to twelve pounds at twelve weeks. Two days after birth the child began to suffer from "blue turns", in which the hands were clenched, the back was arched and the limbs stiffened, and he appeared to lose consciousness. In the first week it had been found necessary to feed him through a pipette and catheter, but gradually his condition improved and he was sucking well at the end of the second week. Breast feeding was continued up to the age of ten months. Physical development was always retarded. The child tried to raise the head at the age of four months, but could not sit up till after twelve months. He could not walk till the age of four years, and even at the time of the meeting he had a poor sense of balance; he was ataxic, with a tendency to lurch towards the right side.

Dr. Southby drew attention to the patient's ataxic gait, strabismus, incoordination and nystagmus. Special investigations, including radiography of the skull, the performance of the Wassermann test and the Casoni test, microscopic examination of the urine and examination of the *fundus oculi*, had shed no light on the cause. Dr. Southby had recently sought the advice of Dr. John F. Williams. Dr. Williams had reported that the patient had a Binet-Simon intelligence quotient of 74%, and thought training was well worth while in a special clinic. Dr. Southby expressed the opinion that the condition was due to birth trauma involving the cerebellum.

DR. J. W. GRIEVE congratulated Dr. Southby on the presentation of such an interesting case. He thought the diagnosis suggested was the most likely one.

Dr. Southby, in reply, excluded Friedreich's ataxia and cerebellar tumour as possible aternatives. He said that he had consulted Dr. Graeme Robertson before the meeting; Dr. Robertson favoured cerebellar agenesis as a basis for the condition, and regarded the prognosis as unfavourable.

Suprarenal Neuroblastoma.

DR. J. W. GRIEVE showed a girl, aged four years, with tumour masses in the skull apparently arising from a primary growth in the left suprarenal medulla. The child's illness had begun with anorexia, languor and fever eight months previously. After four months pallor was noticed and a painless lump appeared in the neck. Lumps later appeared in the left parietal and frontal regions, the mandible and the bridge of the nose, and they had gradually increased in size. Six weeks before the meeting the child had developed a cough; for two weeks the abdomen had been swollen and œdema of the ankles had been present for two days. The patient had bilateral exophthalmos and strabismus. Tumours were easily discernible in the head and neck. There were signs of left pleural effusion, which had been verified by aspiration, uniformly blood-stained fluid being obtained. The abdomen was distended and masses were present in both iliac fossæ. The legs were œdematous. On examination of the blood the hæmoglobin value was found to be 34%. The erythrocytes numbered 1,680,000 and the leucocytes 14,000 per cubic millimetre. A differential leucocyte count gave the following results: polymorphonuclear cells 55%, lymphocytes 32%, metamyelocytes 6%, myelocytes 2%, mononuclear cells 3%, eosinophile leucocytes 3%. On microscopic examination the urine was clear, and skiagrams revealed secondary deposits in the skull, the humerus and both femora.

Dr. Grieve thought the condition hardly fitted in with either the Pepper or the Hutchinson syndrome. His review of the literature led to the conclusion that the matter was best summed up in Dr. Reginald Webster's "Pathological Reports from the Children's Hospital". Dr. Grieve had never previously seen secondary deposits in the mandible.

DR. REGINALD WEBSTER said that he had had an opportunity of carrying out biopsy investigation of a gland removed from the patient's neck, but the findings were inconclusive. He pointed out that metastasis in the skull did not exclusively point to the left suprarenal gland as the original site of the primary, nor did hepatomegaly necessarily indicate a primary origin in the right suprarenal.

Dr. Grieve, in reply, quoted a case he had encountered many years earlier in an adult. That patient had presented with a lesion in the hip joint and later had suffered from heart block.

Multiple Chronic Ulcers of the Neck and Arms.

DR. HOWARD WILLIAMS showed a boy, aged eleven years, who had developed abscesses, incision of which left chronic ulcers. Although he was previously perfectly healthy, the boy had been ill for six months at the time of the meeting. The illness had commenced with moderately severe faucial diphtheria, for which he had been treated in the usual way with antitoxic serum at the Queen's Memorial Infectious Diseases Hospital, Fairfield. Three days after the onset a swelling had developed on the lower portion of the right jaw and had increased rapidly in size, affecting the subcutaneous tissues over the cheek, ear and neck. When it retrogressed, a hard, tender mass was left, which softened after three weeks. The abscess was incised and thick pus was obtained, a smear from which yielded no growth of organisms in nutrient media, and no organisms were found in specially stained slides. Unsuccessful attempts were made to grow actinomyces and *Mycobacterium tuberculosis*. Other abscesses of a similar nature formed later on the left side of the neck and were incised. All the incisions failed to heal, and the boy had had several transfusions of blood

on account of severe secondary anæmia. Early in June, 1941, he was admitted to the Children's Hospital with seven chronic ulcers on the neck and left arm, and since then several low-grade inflammatory lesions had appeared on the arms. He was moderately well nourished, but the ulcers were indolent and the edges were undermined. One of the ulcers was at the site of a transfusion wound. There was a slight sero-sanguinous discharge from the right nostril, which was excoriated, and there was a small inflammatory nodule on the right cheek.

Dr. Williams said that in skiagrams of the chest, mandible and cervical portion of the spine it was obvious that the bones were not involved and there were no deposits from a primary source in the chest. Diphtheria organisms had been cultured from a nasal swab, but samples of the blood and pus had been submitted for culture without success. Even Dr. Webster had not been able to grow tubercle bacilli, and inoculation of a guinea-pig had failed to provide any evidence of tuberculous infection. The blood serum had not yielded the Wassermann reaction. The hæmoglobin value was estimated at 51%; the erythrocytes numbered 3,270,000 and the leucocytes 5,100 per cubic millimetre, and the differential leucocyte count gave results within normal limits.

Dr. Williams said that on May 30 some tissue from the edge of an abscess had been submitted for the opinion of Professor MacCallum, who had reported that the process suggested neoplastic infiltration, possibly of lymphoid tissue origin, corresponding in some measure to the lesions which had been described as "sarcoids". On June 13 Dr. Webster examined a similar biopsy specimen and reported that the material was vascular pyogenic granulation tissue; there was no evidence of tuberculous or other specific ætiology.

Dr. Williams added that the Mantoux test with the usual dilutions had been carefully carried out, but no reaction had been obtained. The ulcers had shown no response to treatment with mercurochrome (5% solution) or to exposure to ultra-violet light. The boy had been taking potassium iodide internally. Dr. Williams thought the members would be interested to hear the history and see the patient. He asked for suggestions concerning the diagnosis and treatment, and thought that Dr. Webster might comment on the pathology.

DR. REGINALD WEBSTER said that he had been much interested in the diagnostic problem presented by the patient. He had received an adequate quantity of pus, subsequently found to be of amorphous character and sterile by routine methods of cultivation. The pus had been aspirated from a recently developed fluctuant swelling and had been found to contain no acid-fast bacilli, no spores and no filaments of actinomycosis. No growth had eventuated after four weeks' incubation in preparations designed for the cultivation of *Mycobacterium tuberculosis*, and a guinea-pig inoculated subcutaneously had not as yet developed a local granuloma or enlargement of inguinal glands. It therefore appeared that the efforts to demonstrate the presence of *Mycobacterium tbuerculosis* would prove unsuccessful.

Dr. Webster said that he understood that, prior to the boy's admission to the Children's Hospital, Professor MacCallum had examined a biopsy preparation and had suggested consideration of a diagnosis of "sarcoids" or the Besnier-Boeck syndrome. Dr. Webster said that his information on "sarcoids" had been gained from a demonstration by Professor C. G. Lambie and Dr. A. H. Tebbutt at a meeting of the Royal Australasian College of Physicians in Sydney, September, 1939. On that occasion he had been impressed with the resemblance to tuberculosis shown by Dr. Tebbutt's histological preparations and by the strong suggestion of Hodgkin's disease in a spleen which Dr. Tebbutt showed as a macroscopic specimen. In a comprehensive article published in THE MEDICAL JOURNAL OF AUSTRALIA of June 15, 1940, Professor Lambie had given a clear description of the histology of the Besnier-Boeck disease, both in the text and by excellent photomicrographs. A cardinal point in the histology of Boeck's "sarcoids" was that caseation and necrosis did not occur. It was therefore highly improbable that a diagnosis of the Besnier-Boeck disease could be supported in the case under discussion, in which softening unconnected with pyogenic infection had occurred in a recent lesion to a degree which permitted the aspiration of its contents.

DR. ROBERT SOUTHBY thought that, since the lesions had developed after diphtheria, it was possible that the ulcers were due to the diphtheria or to the treatment given. One occasionally encountered persons susceptible to serum; those lesions were possibly reactions to the serum given for the diphtheria. Dr. Southby also wondered whether the association of severe toxæmia and nutritional disturbance, especially vitamin lack, might have set the process in motion. In any case, a diet containing adequate supplies of vitamins and iron was indicated. In spite of the absence of reaction to the Wassermann test, a trial of treatment with arsenic would be worth while.

DR. KATE CAMPBELL thought that the lesions might be a manifestation of the severe allergic reaction which occasionally followed the administration of serum.

DR. M. O. KENT HUGHES said that, since a tuberculous origin was not excluded, a course of ultra-violet irradiation would be worth a trial.

Dr. Williams, in reply, said that one could argue from the extensive negative findings that the lesions were probably not due to any known toxic agent; therapy therefore must be empirical. He appreciated Dr. Southby's suggestions. He informed Dr. Kent Hughes that ultra-violet light had been tried without success. The ulcers clinically gave the impression of being tuberculous in nature.

Cerebral Abscess.

DR. REGINALD WEBSTER showed a specimen from the brain of a boy, aged twelve years, in which was seen a large cerebral abscess, occupying the right temporo-sphenoidal lobe and extending anteriorly and posteriorly to invade the frontal and occipital lobes respectively. The lateral ventricle on the right side was distorted and narrowed, that on the left side considerably dilated. The immediate cause of death was purulent basal meningitis. Mucopus was present in both middle ear chambers, and in the absence of trauma, pyæmia, bronchiectasis or other pulmonary suppuration, it was to be presumed that the abscess in the right half of the cerebrum was a sequela of chronic suppurative *otitis media*. The autopsy, however, revealed no evidence of direct spread of infection to the cerebral substance in the form of erosion of the *tegmen tympani*, subdural granulation tissue or abscess.

Dr. Webster said that the boy had been admitted to hospital in an unconscious condition, with the provisional diagnosis of cerebral tumour, based on a letter from his private medical attendant. It was not necessary to give the neurological findings in detail, but Dr. Webster mentioned the fact that severe bilateral papilloedema had been present. Eight days after the boy's admission to the Children's Hospital Dr. H. Douglas Stephens undertook ventriculography. He had been unable to aspirate any fluid from the right lateral ventricle, but had withdrawn blood-stained cerebro-spinal fluid from the ventricle of the left side. Air was introduced into the ventricular system via the left ventricle. The boy died four days later.

The child's clinical history indicated that he had been deaf and dumb since birth, but had maintained good health until two years earlier, when he began to suffer from convulsive seizures. The seizures occurred at first at long intervals, but during the last two months they had increased in frequency and severity. Three weeks prior to his entering the Children's Hospital the boy had developed acute tonsillitis; he had been confined to bed ever since the onset of the throat infection.

Dr. Webster asked, in view of the proverbial capacity for chronicity displayed by cerebral abscesses, whether it was possible that this abscess had existed for the two years during which the boy had been subject to convulsive seizures. The anatomical features and character of the pus were consistent with a chronic process. Boyd had recorded the story of a soldier who sustained a head wound in the war of 1914-1918. Death two years later was the result of the rupture into the ventricular system of a small frontal lobe abscess. In the interval the only disturbances of health had been attacks of headache, dizziness on stooping and occasional convulsive seizures. It seemed possible, if not probable, that the measures involved in ventriculography had precipitated the rupture of the abscess into the lateral ventricle, with consequent purulent meningitis.

Glomerulo-Nephritis.

Dr. Webster next showed the kidneys of a girl, aged nine years, who had recently died of acute nephritis. The diagnosis was clear from the moment of the child's admission to hospital; it was also apparent that renal failure was impending. The girl was pallid and œdematous, complained of severe headache and vomited frequently. Hæmaturia, the presence of numerous casts in the urine, and a degree of nitrogen retention, represented by a blood urea content of 150 milligrammes per 100 cubic centimetres, were contributory findings in a complete clinical picture. It had not been possible to obtain precise information concerning the antecedents of the girl's illness, as her mother was a cripple and the deaconess who brought the patient to the hospital had no knowledge of facts or circumstances likely to be of importance in relation to the illness. There had been an indefinite statement about a sore throat, and another, more positive, to the effect that the child had suffered from sores on the body for some time. Existing lesions of impetigo were in fact to be observed on the legs and on the right forearm. An attack of pulmonary œdema was the immediate

precursor of death, forty-eight hours after the child's admission to hospital, an interval during which she displayed alternate delirium and coma.

Dr. Webster said that he did not propose to discuss the morbid anatomy of glomerulo-nephritis, or to detail the post-mortem findings; his object in presenting the kidneys was to induce discussion on ætiological factors in glomerulo-nephritis. In connexion with the impetigo from which this girl had evidently suffered for some time, Dr. Webster recalled that Fishberg, in his book "Hypertension and Nephritis", had stated that acute glomerulo-nephritis could be associated with recent infection—sore throat, scarlet fever, skin infection or pneumonia—in 84% of instances. Fishberg allotted skin infection a share of 8% in the ætiology of the disease. It was a truism to say that acute glomerulo-nephritis was intimately related to recent streptococcal infection, most frequently of the throat, and it would be trite and superfluous to dwell on its association with scarlet fever. For many years the direct action of circulating streptococcal toxin on the glomeruli was accepted as good and sufficient cause for the renal complication of scarlet fever, although admittedly it was difficult to account for the clinical observations that the onset of nephritis was usually delayed until the patient appeared to be entering on convalescence, and that the incidence of nephritis bore no relation to the severity of the attack. To cover these facts the thesis had been formulated that glomerulo-nephritis was the expression of an allergic reaction.

Dr. Webster said that he was prepared to be considered conservative, although he hoped that he had not yet reached the "fixed period" described by Sir William Osler in one of his delightful and scholarly "Aequanimitas" addresses. But he found it difficult to accept much of the allergy which had pervaded medical literature to the point of supersaturation during recent years. The demonstration of the importance of allergic phenomena in the response to tuberculous infection seemed to have initiated a vogue under which an increasing number of diseases was to be placed on an allergic basis. Among these were diseases of no less importance than acute rheumatism, lobar pneumonia and glomerulo-nephritis. Dr. Webster regarded the extension of the allergic hypothesis to cover glomerulo-nephritis as an as yet unjustified attempt to surmount a difficulty in ætiology. As he understood the conception, the acquisition of immunity by the patient involved a concomitant hypersensitiveness on the part of the tissues to the bacterial protein or the toxin of the streptococcus; the renal tissue participated in the general allergic state and responded with an exaggerated reactivity, should the sensitizing agent—streptococcus disintegration product or toxin—subsequently lodge in the glomerular filter. There was an implied assumption in this hypothesis that a state of allergy was inseparable from that of immunity—a belief which had been challenged, and as far as he was capable of assessing the position, successfully contested by Arnold Rich. Rich maintained that allergy and immunity were two distinct and separate phenomena, which had been confused simply because they frequently coexisted. He had further stated that in spite of widespread and dogmatic assertions about the protective role of allergy, there was not one single observation or experiment on record through which hypersensitiveness had been shown to be necessary for the operation of immunity in any infection under any condition whatsoever.

Dr. Webster said that in his view it had yet to be shown on grounds more substantial than theoretical speculation that the tissues, and particularly the renal tissues, of convalescents from scarlet fever reacted in a hypersensitive manner to the streptococcus and its products. The fact that all became "Dick-negative" at an early stage of the disease, both nephritics and those who escaped nephritis, seemed to show that such patients could not be supposed to react in any peculiar way to the streptococcus toxin.

Observations by Friedemann and Deicher were on record, which showed that in cases of scarlet fever complicated by nephritis there was a much more rapid development of bactericidal antibody in the blood than occurred in cases in which nephritis did not supervene; these findings had been obtained by protection experiments with patients' serum and streptococcus culture in mice. It had been suggested that the rapid elaboration of immune bodies led to the liberation of an excessive amount of streptococcus endotoxin. If such were the process culminating in nephritis as a complication of scarlet fever, it was in no sense an allergic reaction.

Numerous experiments had been undertaken to show that glomerulo-nephritis might be the expression of an antigen-antibody reaction. Much of the experimental observation had been made under artificial conditions, worlds apart from anything conceivable in the naturally occurring disease. There was, for instance, the work of Masugi, in whose original experiments rabbits were given repeated injections of a suspension of rat kidney for the purpose of elaborating a "nephrotoxic serum". The serum of such rabbits when injected into rats induced acute inflammatory glomerular lesions. In later work Masugi administered suspensions of rabbit kidney by repeated intraperitoneal injection to ducks, and subsequently injected the serum of ducks so treated intravenously into rabbits. The rabbits undoubtedly suffered an acute glomerulo-nephritis, displayed albumin and casts in the urine, rise of blood pressure, and nitrogen retention, and died of renal failure. Subsequent histological examination of the kidneys revealed a microscopic morbid anatomy identical with that of naturally occurring acute glomerulo-nephritis in human beings. Another form of experiment had been to inject horse serum into the renal artery; this procedure was said to cause a diffuse glomerulitis in animals previously sensitized to horse serum. Of a piece with such experiments of Masugi was that of Long and Finner, who exposed the renal artery in pigs and injected a large volume of fluid containing a specially prepared tuberculin protein. In untreated animals this was without effect on the kidney, but in pigs previously inoculated with tubercle bacilli, which hence were in an allergic state, the result was glomerulo-nephritis.

Dr. Webster said that in his opinion experiments of this type carried no conviction; at most they showed that glomerulo-nephritis might be induced in hapless animals rendered allergic and subjected in their hypersensitive state to an experimental *Blitzkrieg*. No more moderate description was appropriate to the introduction of powerful toxin or concentrated foreign protein directly into the renal artery.

The experimental work of Duval and Hibbard, like that of Friedemann and Deicher to which Dr. Webster had made earlier reference, suggested that when nephritis followed the development of an immune state, the renal lesion was due to an endotoxin liberated by the lysis of surviving bacteria. Rabbits highly immunized with scarlet fever streptococci received an intraperitoneal injection of 50 cubic centimetres of a suspension containing large numbers of living streptococci. In the fluid which could be recovered two hours later these microorganisms had been destroyed, but when the fluid was injected intravenously into other rabbits it produced acute fatal glomerulitis. Dr. Webster expressed the opinion that such an experimental result was not an allergic phenomenon. The lysis of streptococci was to be attributed to the active immunity induced in the animals, for the operation of which an allergic state was in no way necessary; thereafter the free streptococcal endotoxins excited glomerular inflammatory changes by direct action.

In conclusion Dr. Webster submitted that acute glomerulo-nephritis in the majority of instances was due to streptococcal infection, most frequently initiated in the throat; the active agent was the streptococcus toxin, which by virtue of its heavy molecular weight was held up by the glomerular filter. That minute amounts of bacterial protein might excite vascular and exudative changes in renal tissue rendered hypersensitive during the patient's acquisition of immunity was a theoretical possibility, to which many had given the credence due to established fact. Dr. Webster felt that to accept the representation of such diseases as lobar pneumonia and glomerulo-nephritis as manifestations of allergy was to offer too ready an allegiance to a prevailing cult, if not to surrender judgement to the fascination of a name.

Post-Graduate Work.

WEEK-END COURSE AT PARRAMATTA, NEW SOUTH WALES.

THE New South Wales Post-Graduate Committee in Medicine announces that in conjunction with the Central Western Medical Association, it will hold a week-end course at Parramatta on Saturday, October 18, and Sunday, October 19, 1941. The programme is as follows.

Saturday, October 18.

(At Parramatta District Hospital.)

2.30 p.m.—"Some Practical Points in Inhalational Anæsthesia": Dr. W. I. T. Hotten.

4 p.m.—(*a*) "Anatomy of the Eye and Orbit from the Clinical Standpoint", (*b*) "Ocular Injuries": Dr. N. McAlister Gregg.

Sunday, October 19.

(At Parramatta District Hospital.)

10 a.m.—"Enotracheal and Endopharyngeal Anæsthesia": Dr. W. I. T. Hotten.

11.30 a.m.—"The Red Eye—Differential Diagnosis and Treatment": Dr. N. McAlister Gregg.
2 p.m.—(*a*) "Intravenous Anæsthesia", (*b*) "Low Spinal Anæsthesia": Dr. W. I. T. Hotten.
3 p.m.—"Cataract and Squint": Dr. N. McAlister Gregg.

The fee for the course will be one guinea, and those intending to be present are requested to notify Dr. K. S. Macarthur Brown, "Brislington", 12, George Street, Parramatta, as soon as possible.

Correspondence.

WHO GROWS THE CABBAGES?

SIR: Dr. Walker's letter on cabbage culture in your issue of August 30 was very welcome, and most timely. Her suggestions for freer and fuller discussion of medical reorganizational problems are, I think, very necessary.

I have often felt a little bewildered at the atmosphere of privacy and secrecy which surrounds the work of British Medical Association Branch councils, and other medical bodies. I can only imagine one good reason for such privacy, namely to allow members to speak freely and to change their opinions in debate without being liable to misrepresentation. That is in a sense reasonable, but should not be allowed to outweigh other considerations.

A great deal of a council's work is humdrum routine, and the decisions are well and wisely made by the members' elected representatives. But even so, there seems no need for the agenda papers to be sent out as "Private and Confidential" to council members. There would, it seems to me, be advantage rather than disadvantage in sending a copy to every member of the Branch.

Similarly, a report from a subcommittee to a council is not helped by being regarded as a confidential document until it has been finally considered by the council and is ready to be put to the Branch. It would surely be in every way better if such a report were given the widest possible publicity among members of the profession before it was presented, while even it was being prepared. Members generally would have had the chance to share, by their expressed opinions, in its formulation, and would not be called together in a general meeting to discuss something entirely new to them, of the principles of which they are ignorant and to which they have been able to give no preliminary thought.

I am not in love with the idea of a formal opposition. It could produce divided loyalties, from which surely we have already suffered enough in *Australia Felix*. And I have never been able to feel that a referendum vote is a good way of reaching important and wise decisions.

What does seem to me to be needed is to regard all members of our profession as vitally interested parties; to keep them informed of movements as they emerge from obscurity, before they even mature; to use the suggestions of such members as are interested enough to give any, in the formation of policies and decisions; and to endeavour to have the debate on which the final decision rests as well informed and intelligent as possible.

For this purpose Dr. Walker's suggestions seem to me to be admissible. If on the grounds of expense they are not immediately practicable, there seems to be no reason why publicity rather than privacy should not be the principle on which the work of councils is based. On a subcommittee being appointed to consider a point of policy, for instance, what objection could there be to notifying all members of the Branch of the formation and purpose of such subcommittee, and giving the postal address of its members, to whom persons interested could send their views?

Yours, etc.,

ARTHUR E. BROWN.

Colac,
Victoria,
September 7, 1941.

MEDICAL SOCIOLOGY.

SIR: Practically all the medical sociologists who have recently been expounding their Utopias both in your columns and the lay Press seem to accept the "inevitability" of the socialization of medicine with the same fatalistic docility as half the world was accepting the "invincibility" of German arms before Russia exploded the fallacy. If I gauge correctly the temper of medical men I meet, the great majority of them are uncompromisingly opposed to any such revolutionary change. But with characteristic Hippocratic reticence they remain inarticulate. Socialization means a levelling down of present standards to mediocrity and, as in ordinary politics, makes its chief appeal to the mediocre. It is a bureaucratic, soul-destroying regimentation intolerable to scientific men of independent free minds and character. The efficiency and high standard of medical service depend incontrovertibly on the professional ability, enthusiasm and integrity of the individuals who perform it—not, as our dreamers and unrealists would have us believe, on the numbers of "satisfied" dupes who imagine they are getting service from so many obliging "rubber stamps". Our high duty is not to try to devise any such scheme in order to placate politicians, who cannot understand the principles involved, nor even to compromise with any such scheme, but by a militant vigilance resist from the beginning any move likely to destroy our present system, which is to medicine what democracy is to civil politics. Let us assert in no uncertain terms that we will fight to the last ditch any attempt to ram down our throats a veritable medical Fascism.

Socialization has not come. It cannot come if we stand firm in our resolve never to lower our standards of service and if we organize to this end whilst there is still time.

Yours, etc.,

R. D. DAVEY.

Croydon Park,
New South Wales,
September 12, 1941.

THE BUCKLAND HOSPITAL, SPRINGWOOD, NEW SOUTH WALES.

SIR: I should like to bring under the notice of the members of our profession the existence of The Buckland Hospital, Springwood, which is a female convalescent hospital.

This is an up-to-date institution. It is not run for profit and is perpetually endowed by Sir Thomas Buckland. The fees are very moderate and within the means of most people.

The hospital is not for mental patients.

It is not being utilized to the extent that one might expect. I feel sure that if these facilities are known to medical practitioners the hospital will be fully availed of.

Yours, etc.,

G. MONCRIEFF BARRON.

Leona,
Manly,
New South Wales.
September 13, 1941.

PREVENTIVE MEDICINE.

SIR: Some public health officers have been talking of preventive medicine recently as though it was the province of the public health officers alone. It has even been said that the general practitioners and specialists are becoming more and more divorced from prevention of disease, and many practitioners are beginning to believe it. It is suggested that if the public health service was greatly extended there would be much more preventive medicine.

In my opinion the private practitioner plays by far the greatest part in preventive medicine.

1. In the main it has been private practitioners who have discovered the cause of diseases. This is surely a big step towards prevention of disease; in fact, the essential step.
2. Preventive medicine for the private practitioner begins before the birth of their patients with antenatal care. Surely this is an important aspect of preventive medicine, about which pages could be written.
3. After birth, supervision by the practitioner is a large factor in preventing the development of rickets, malnutrition and spread of childhood infections *et cetera*. Surely care of illness by the practitioner in infancy and childhood prevents the development of malnutrition and chronic ill health.
4. Vaccination against smallpox, diphtheria, typhoid *et cetera* is advised and carried out by the private practitioner.
5. Care of wounds by the private practitioner prevents added infection, cellulitis, septicæmia, tetanus.
6. Prevention of puerperal sepsis is carried out by the private practitioner.
7. Nearly all infectious diseases are diagnosed, and the prevention of spread of infection supervised, by general practitioners.
8. Advice *re* marriage and child-bearing in tuberculosis, epilepsy and mental disease.
9. Prevention of asthma, hay fever *et cetera* by treatment.
10. Prevention of spread of syphilis and gonorrhœa.

Is not the prevention of death by treatment a sort of preventive medicine?

I know the public heath departments do excellent work in connexion with a small branch of preventive medicine, but I am convinced that a very large part indeed of preventive medicine is carried out by the private practitioner.

I hope this letter may stimulate thought on this matter. It would be a good thing too if all practitioners would think out for themselves whether they think a large increase of public health officers, leading to nationalization of medical practice, is or is not likely to be for the benefit of the public as patients.

Yours, etc.,

G. C. WILLCOCKS.

143, Macquarie Street,
Sydney,
September 13, 1941.

Naval, Military and Air Force.

ROYAL AUSTRALIAN AIR FORCE AIR AMBULANCE UNIT.

MEMBERS of the medical profession will be interested to learn that as a result of work carried out during the last few months, Number 1 Royal Australian Air Force Air Ambulance Unit was formed, equipped and flown from Australia to the Middle East, where the ambulances are now in commission.

The work of converting the aircraft, which had been made available for the purpose of air transport of casualties, was carried out in Australia from designs drawn up by the Royal Australian Air Force Medical Directorate, in which the Director of Medical Services, Royal Australian Air Force (Air-Commodore Hurley), was assisted by Squadron-Leaders S. F. Reid (now overseas), W. M. Borland and G. Simpson, the last mentioned having had much experience in the use of aircraft in the Australian Inland Medical Services. Technical officers of the Royal Australian Air Force were also made available for the work, which was carried out entirely by Australian craftsmen.

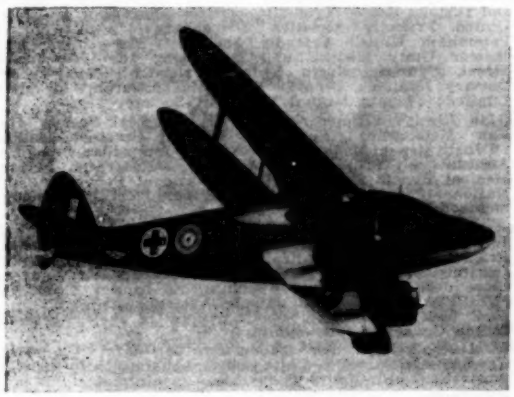

FIGURE I.

Accommodation is provided in each aircraft for six patients on stretchers and two sitting patients as well as for the crew. Special light-weight metal tubular stretchers were devised, and numerous special fittings, including the provision of oxygen supply and equipment to deal with emergencies *en route*, have been provided.

The personnel of the unit consists of a complete Royal Australian Air Force maintenance staff, including fitters, riggers, wireless operators *et cetera*, as well as air crew and nursing orderlies.

The maintenance and ground personnel proceeded abroad by sea, while the equipped aircraft, with medical personnel, flew from Melbourne to the Middle East, where they arrived safely. After mechanical overhaul the aircraft were then put into service.

The disposal and actual details for the employment of the unit were determined at a conference of officers held at Headquarters, Middle East. Those present at the conference included the Director of Medical Services of the British Forces, the Director of Medical Services, Australian Imperial Force (Major-General Burston), the Principal Medical Officer, Royal Air Force, Middle East, and Group-Captain Daley, Deputy Director, Royal Australian Air Force Medical Service. The last mentioned accompanied the unit from Australia.

FIGURE II.

The unit is primarily for the use of Australian personnel, but may be used when needed for others at the discretion of the Australian Imperial Force administrative medical officer controlling its movements.

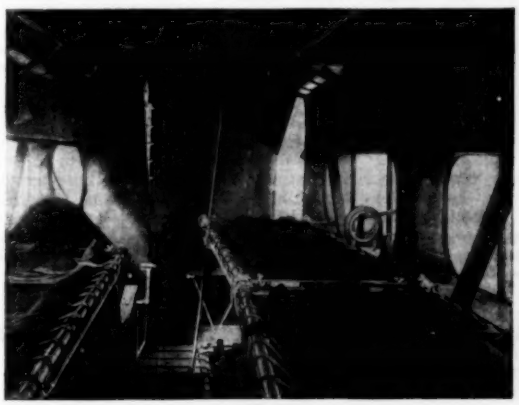

FIGURE III.

The medical authorities in the Middle East, and also in the other places *en route* where the aircraft were inspected, feel that such a means of rapid and comfortable transport offers a real contribution towards the problem of providing early and effective treatment of serious casualties.

The fitting of other aircraft equipped on similar lines is also in progress for Australian requirements.

NOTICE.

THE Deputy Director of Medical Services of the Eastern Command wishes to draw the attention of officers on the active list and on the reserve of officers to a meeting of the Section of Preventive Medicine of the New South Wales Branch of the British Medical Association which will be held at the Robert H. Todd Assembly Hall, British Medical Association House, 135, Macquarie Street, Sydney, on Thursday, October 2, 1941, at 8.15 o'clock p.m. The subject for discussion will be cerebro-spinal meningitis. Dr. W. C. Sawers will read a paper on the epidemiology of the condition, and Dr. N. J. Symington will read one on diagnosis and treatment. It is hoped that as many officers as possible will attend.

CASUALTIES.

ACCORDING to the casualty list received on September 22, 1941, Captain R. A. Playoust, A.A.M.C., of Mosman, New South Wales, and Captain L. P. Sapsford, A.A.M.C., of Allora, Queensland, previously reported missing, are now reported prisoners of war.

MEDICAL WAR RELIEF FUND.

THE following is a seventeenth list of contributions to the Medical War Relief Fund established by the Federal Council of the British Medical Association in Australia for the relief of distressed medical practitioners in Great Britain.

New South Wales.

£5 5s.: Dr. J. S. MacMahon, Dr. K. Kirkland, Dr. W. R. Page, Dr. R. O. Williams (second contribution).
£3 3s.: Dr. A. E. Harker, Dr. H. T. C. MacCulloch, Dr. G. T. Hunter.
£2 2s.: Dr. W. J. R. Nickson, Dr. N. McA. Gregg, Dr. C. G. Crawford, Dr. C. V. Roper, Dr. H. R. Sear, Dr. L. J. Shortland, Dr. L. H. Hughes, Dr. J. Ross.
£1 1s.: Dr. R. K. Burnett, Dr. J. A. L. Wallace, Dr. H. K. Porter, Dr. H. J. Daly.

Western Australia.

£1 1s.: Dr. W. A. Hillman.

Nominations and Elections.

THE undermentioned has applied for election as a member of the New South Wales Branch of the British Medical Association:

Duncan, Ian Lovell, M.B., B.S., 1940 (Univ. Sydney), NX35135, Captain, A.A.M.C., Headquarters, Royal Australian Engineers, 8th Australian Division, Overseas.

Obituary.

ERNEST ALBERT WHERRETT.

WE regret to announce the death of Dr. Ernest Albert Wherrett, which occurred on September 22, 1941, at Ashfield, New South Wales.

Australian Medical Board Proceedings.

NEW SOUTH WALES.

THE undermentioned have been registered, pursuant to the provisions of the *Medical Practitioners Act*, 1938-1939, as duly qualified medical practitioners:

Andrews, James Campbell, M.B., Ch.B., 1940 (Univ. Edinburgh), Lily Bank, Wauchope.
Mills, Margaret Mary, M.B., Ch.B., 1922 (Univ. Aberdeen), 10 Mackenzie Street, Lindfield.
Thomson, Charles Hutcheon, M.B., Ch.B., 1935 (Univ. Edinburgh), c.o. British Phosphate Commissioners, 16, Spring Street, Sydney.

Books Received.

"The Medical Annual: A Year Book of Treatment and Practitioner's Index", edited by H. L. Tidy, M.A., M.D., F.R.C.P., and A. R. Short, M.D., B.S., B.Sc., F.R.C.S; 1941. Bristol: John Wright and Sons Limited. Demy 8vo, pp. 561, with illustrations. Price: 20s. net.

"A Textbook on the Nursing and Diseases of Sick Children", edited by A. Moncrieff, M.D., F.R.C.P.; Third Edition; 1941. London: H. K. Lewis and Company Limited. Demy 8vo, pp. 654, with 142 illustrations. Price: 21s. net.

"Massage and Medical Gymnastics", by M. V. Lace, with a foreword by J. Mennell, M.A., M.D., B.C.; Second Edition; 1941. London: J. and A. Churchill Limited. Demy 8vo, pp. 250, with 120 illustrations. Price: 12s. 6d. net.

"The Student's Pocket Prescriber and Guide to Prescription Writing", by D. M. Macdonald, M.D., D.P.H., F.R.C.P.E.; Eleventh Edition; 1914. Edinburgh: E. and S. Livingstone. Demy 18mo, pp. 328. Price: 3s. 6d. net.

"First Aid Through Photographs", by L. S. Michaelis, M.D.; photographs by V. Elkan; 1941. London: Longmans, Green and Company. Crown 8vo, pp. 64, with illustrations. Price: 2s. net.

Diary for the Month.

OCT. 1.—Western Australian Branch, B.M.A.: Council.
OCT. 1.—Victorian Branch, B.M.A.: Branch.
OCT. 2.—South Australian Branch, B.M.A.: Council.
OCT. 3.—New South Wales Branch, B.M.A.: Annual Meeting of Delegates.
OCT. 3.—Queensland Branch, B.M.A.: Branch (Ordinary).
OCT. 7.—New South Wales Branch, B.M.A.: Council (Quarterly).
OCT. 10.—Queensland Branch, B.M.A.: Council.
OCT. 14.—Tasmanian Branch, B.M.A.: Branch.
OCT. 15.—Western Australian Branch, B.M.A.: Branch.
OCT. 22.—Victorian Branch, B.M.A.: Council.
OCT. 24.—Queensland Branch, B.M.A.: Council.
OCT. 30.—New South Wales Branch, B.M.A.: Branch.
OCT. 30.—South Australian Branch, B.M.A.: Branch.
OCT. 31.—Tasmanian Branch, B.M.A.: Council.

Medical Appointments: Important Notice.

MEDICAL PRACTITIONERS are requested not to apply for any appointment mentioned below without having first communicated with the Honorary Secretary of the Branch concerned, or with the Medical Secretary of the British Medical Association, Tavistock Square, London, W.C.1.

New South Wales Branch (Honorary Secretary, 135, Macquarie Street, Sydney): Australian Natives' Association; Ashfield and District United Friendly Societies' Dispensary; Balmain United Friendly Societies' Dispensary; Leichhardt and Petersham United Friendly Societies' Dispensary; Manchester Unity Medical and Dispensing Institute, Oxford Street, Sydney; North Sydney Friendly Societies' Dispensary Limited; People's Prudential Assurance Company Limited; Phœnix Mutual Provident Society.

Victorian Branch (Honorary Secretary, Medical Society Hall, East Melbourne): Associated Medical Services Limited; all Institutes or Medical Dispensaries; Australian Prudential Association, Proprietary, Limited; Federated Mutual Medical Benefit Society; Mutual National Provident Club; National Provident Association; Hospital or other appointments outside Victoria.

Queensland Branch (Honorary Secretary, B.M.A. House, 225, Wickham Terrace, Brisbane, B.17): Brisbane Associated Friendly Societies' Medical Institute; Bundaberg Medical Institute. Members accepting LODGE appointments and those desiring to accept appointments to any COUNTRY HOSPITAL or position outside Australia are advised, in their own interests, to submit a copy of their Agreement to the Council before signing.

South Australian Branch (Honorary Secretary, 178, North Terrace, Adelaide): All Lodge appointments in South Australia; all Contract Practice appointments in South Australia.

Western Australian Branch (Honorary Secretary, 205, Saint George's Terrace, Perth): Wiluna Hospital; all Contract Practice appointments in Western Australia.

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